Transactions

of the

Forty-First Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

NEW YORK CITY, NEW YORK

September 27 to October 3, 1936

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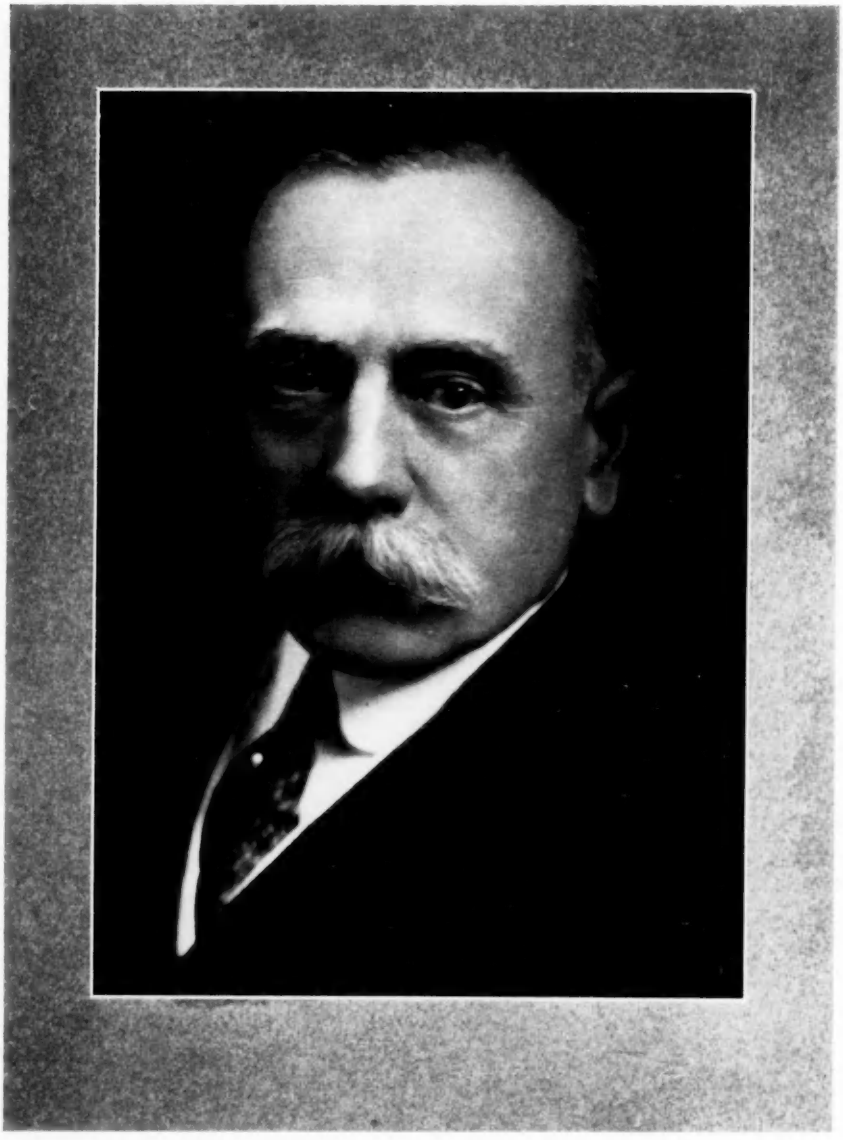


Herbert Stanley Birkett

C.B., V.D., M.D., C.M., LL.D.

* * *

Dr. H. S. Birkett was born July 17, 1864, in Hamilton, Ontario, his father being English by birth and his mother Canadian. He studied medicine at McGill University, Montreal, graduating M.D., C.M., in 1886. The next two years were spent in postgraduate training in otolaryngology in Vienna, Berlin, Paris and London.

In 1889, Dr. Birkett was appointed to the Montreal Dispensary, and in the following year to the Montreal General Hospital; subsequently he was attached to other Montreal hospitals of more recent foundation — the Royal Victoria, the Alexandra Hospital for Infectious Diseases, and the Children's Memorial. The year 1889 marked the beginning of his long teaching connection with McGill University, first as Senior Demonstrator in Anatomy and later as Professor of Otolaryngology for thirty-six years. He served as Dean of the Faculty of Medicine of McGill University from 1914 till 1921, and on retiring from this office received the honorary degree of LL.D.

Dr. Birkett is a Fellow of the Royal Society of Medicine of London, England, of the American College of Surgeons, and of the Royal College of Physicians and Surgeons of Canada. He is a member of the British Medical Association and the Canadian Medical Association. He is an Honorary Member of the American Academy of Ophthalmology and Otolaryngology, and of the Scottish and Italian Oto-Laryngological Societies, and a member of the International Collegium of Otolaryngology. He represented Canada at the International Congress of Oto-Laryngology in Berlin, Germany, in August, 1936. He has been President of the American Laryngological Association, the American Otological Society, and the American Laryngological, Rhinological and Otological Society. He was Honorary President of the Clinical Congress of Laryngology held in London, England, in 1919. He has acted as Vice-President of the Section of Otology and Laryngology at four annual meetings of the British Medical Association — in 1897, 1906, 1930 and 1932 — and was Vice-President of the Pan-American Medical Congress held in Brazil in 1935. He is a past-President of the Montreal Medico-Chirurgical Society and is now Honorary President of the Oto-Laryngological Section. He is Honorary President of the MacKay Institute for Deaf-Mutes in Montreal.

For fifty years, Dr. Birkett has been a medical officer in the Militia of Canada. In 1914, at the outbreak of the Great War, he organized No. 3 Canadian General Hospital (McGill), and remained its commanding officer until 1918. He was then promoted to the rank of Brigadier-General, with the official title of Assistant Director General of Medical Services for the Overseas Military Forces of Canada. He served as Consultant in Oto-laryngology for the Boulogne Area in France from 1915 to 1918.

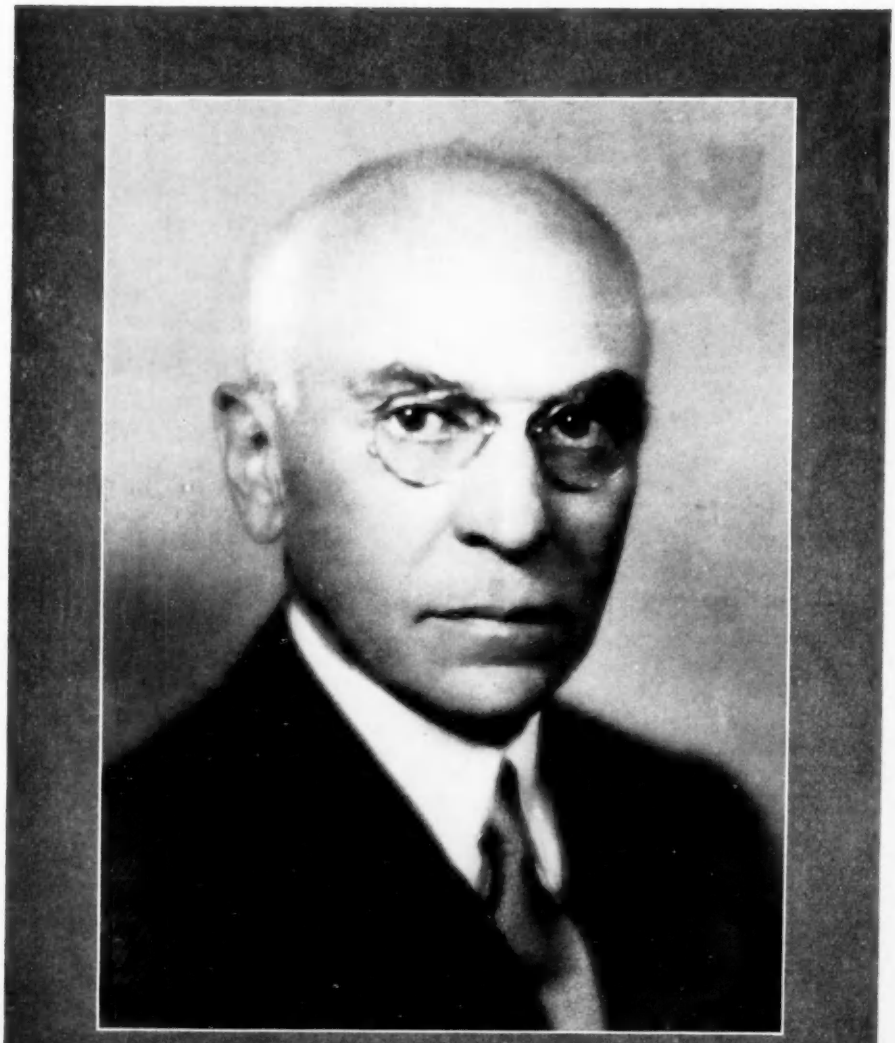
Dr. Birkett is the author of some forty contributions to medical literature. He wrote on "Diphtheria" for Hare's System of Therapeutics *(1890), on "Syphilis of the larynx, trachea and bronchi" in Buck's* Handbook of Medical Science *(1902), and on "Diseases of the oro- and naso-pharynx" in the textbook of Posey and Wright (1902). He contributed a chapter on "Diseases of the larynx" for Osler's* System of Medicine, *and on "Diseases of the tonsil" for Jackson and Coates' book (1929).*

Dr. Birkett has addressed many societies, among others the British Medical Association, by invitation, on "Vasomotor rhinitis" (1910) and on "The education of the specialist in laryngology and otology" (1912), the Semon Lecture at the University of London in 1922.

* * *

The Academy appreciates the honor of dedicating this issue of the Transactions to

GENERAL HERBERT STANLEY BIRKETT



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1917	Dr. W. L. Dayton Lincoln, Nebr.	Dr. Edward Stieren Pittsburgh, Pa.	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Pittsburgh
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1926	Dr. E. C. Ellett Memphis, Tenn.	Dr. Austin A. Hayden Chicago, Ill.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Colorado Springs
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1928	Dr. Luther C. Peter Philadelphia, Pa.	Dr. Frank L. Dennis Colorado Springs, Colo.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	St. Louis
1929	Dr. Harris P. Mosher Boston, Mass.	Dr. Frank E. Burch St. Paul, Minn.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	Atlantic City
1930	Dr. William H. Wilder Chicago, Ill.	Dr. Frank R. Spencer Boulder, Colo.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	Chicago

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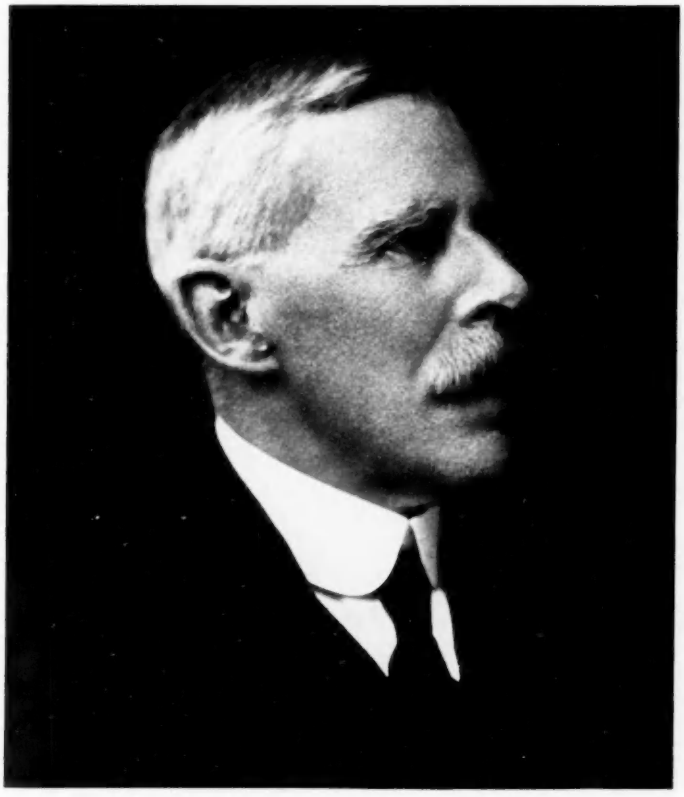
1933—Dr. Burt R. Shurly	Detroit, Mich.	Dr. Nelson Miles Black Miami, Fla.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	Boston
1934—Dr. John M. Wheeler	New York, N. Y.	Dr. LeRoy A. Schall Boston, Mass.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	Chicago
1935—Dr. Wells P. Eagleton	Newark, N. J.	Dr. W. Thornwall Davis Washington, D. C.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	Cincinnati
1936—Dr. Frank E. Burch	St. Paul, Minn.	Dr. Gordon F. Harkness Davenport, Iowa	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	New York

The Guest of Honor



SIR JOHN HERBERT PARSONS, D.Sc.
F.R.C.S., F.R.S.
LONDON, ENGLAND

Joint Session

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Joint Session

The American Academy
of Ophthalmology and Otolaryngology

Forty-First Annual Meeting
New York • 1936

ADDRESS OF THE PRESIDENT

* * *

TRAINING FOR OPHTHALMOLOGY AND OTOLARYNGOLOGY

FRANK E. BURCH, M.D.

SAINT PAUL

MR. CHAIRMAN, Honored Guest, and Members of the Academy: In opening this Annual Meeting, may I take this opportunity to thank the Council and members of the Academy for the high honor you have shown me in electing me to the Presidency. It is an office in which anyone may take pride, for it has been held by many distinguished predecessors. I sincerely appreciate this honor. It has been a rare privilege to work with your secretaries, especially to work with your indefatigable Dr. Wherry, and the New York Committees, all of whom have made *my* task relatively easy. I thank you all.

On behalf of the officers and the Academy, may I extend a welcome to those of you who are new members and visitors. It is my sincerest wish that you may be filled with enthusiasm, that you may acquire the spirit of good fellowship which will pervade at this as it has at every previous meeting.

* * *

ONE OF THE traditions of every leading medical society is that there be a President's address delivered at the opening of its annual conclave. With no idea of encroaching upon the program of the Teachers' Section, I have chosen for my subject a discussion of certain phases of training for ophthalmology and otolaryngology, including future possibilities and opportunities.

The American Academy of Ophthalmology and Otolaryngology has done, and will continue to do, pioneer work in creating and raising standards of specialization. This has been accomplished by excellent programs and instructional courses, by surveys, study and discussion of methods of graduate training by its Teachers' Section, and by its support of the Special Boards

for certification. One of our important functions is the fostering and encouragement of every type of organized teaching for the production of well-trained, efficient, safe practitioners of our specialties.

Medical knowledge is not static. Each year many new discoveries add to or modify previous knowledge. Merely to keep apace with progress in one's own field demands concentrated interest. As a single example, consider the role of electricity in the advances made during the last few decades. Roentgenoscopy, ophthalmoscopy, transillumination, bronchoscopy, biomicroscopy, and other means of viewing vital pathology have made diagnosis and prognosis more accurate, therapy more effective. Continued contributions to biochemistry and physiology and pathology permit us to carry out unheard of, undreamed of procedures. The difficulties and complexities which arise from this constant expansion of medical science have increased the necessity for more fundamental and basic preparation for practice of even a single branch of medicine.

ADVICE TO CANDIDATES FOR TRAINING

We who are established in practice have a definite duty and important influence in the matter of selecting and advising the candidate for training in ophthalmology and otolaryngology. We attempt an impersonal rating of his personality and character, motives and special aptitude, and his actual ability to attain proficiency. We inquire into his preliminary training.

A few years of general practice often provide an excellent beginning, although a longer period of interne experience may be more valuable. It is expected of the candidate who has not been engaged in private practice that he shall have had a medical or surgical rotating interneship in a general hospital; or, if in a special hospital, one that is part of a medical group. Interneship in an isolated special hospital does not provide sufficient contact with practitioners in other fields of medicine.

He comes to us for advice. First of all, let us impress him with the time factor; that there is no shortcut to efficiency. Older men, coming from general practice, too often begrudge the time, the change in social environment, but are usually able and willing to meet the expense of graduate instruction. Younger men, as a rule, have plenty of time and relatively low earning power, and can afford to acquire training slowly. We stress the importance

of general medical and surgical diagnosis, of the relationship of neurology to ophthalmology and otolaryngology, of thorough acquaintance with the general principles and technical procedures of general surgery, and knowledge of general pathology. From our experience, we emphasize the necessity of a liberal background; explain that knowledge of special anatomy and physiology, biochemistry and special pathology must go far beyond that provided in ordinary undergraduate study. We try to impress the would-be specialist that his success, which means his happiness, will in large measure depend upon the breadth and depth of his training.

FUTURE REQUIREMENTS

The Council on Medical Education is demanding the following requirements, beginning with 1938:

(1) "A period of study after the interneship of not less than three years in clinics, dispensaries, hospitals or laboratories recognized by the Council as competent to provide a satisfactory training in the special field of study.

(2) "This period of specialized preparation shall include:

(a) "Intensive graduate training in anatomy, physiology, pathology and other basic medical sciences, which are necessary to the proper understanding of the disorders and treatment involved in the specialty in question.

(b) "An active experience of not less than eighteen months in hospital clinics, dispensaries or diagnostic laboratories recognized by the Council as competent in the specialty.

(c) "Examinations in the basic medical sciences of a specialty, as well as in the clinical, laboratory and public health aspects.

(3) "An additional period of not less than two years of study and/or practice."

This approximately parallels the requirements of Denmark, Germany and other European countries by endeavoring to prescribe or at least to encourage the amount of training necessary for recognition in each specialty.

This program proposed by the Council has not heretofore been feasible. It is not so even at present. From a practical standpoint, under present conditions, if we are to follow a three-year

educational program, allowing a sufficient period for advanced study of fundamentals, provision must be made for basic instruction during the interneship, residency or fellowship tenure. This arrangement for combined pre-clinical and clinical training seems necessary at present in order that the candidate may not, for economic or other reasons, restrict his basic training, and may properly correlate basic with what would otherwise be purely clinical opportunity. In most instances, there is no alternative arrangement.

OPPORTUNITIES

What are his opportunities? In a recent list of institutions in the United States approved by the Medical Council of the American Medical Association for 1935 are found 292 available internships or residences for training in ophthalmology and otolaryngology. In 36 institutions, of which 11 are either special hospitals or special departments of general hospitals, there are 95 opportunities for training in ophthalmology. Forty institutions, of which 10 are special hospitals or special departments of general hospitals, offer 112 positions for training in otolaryngology. Either combined or separate training in eye, ear, nose and throat provides 85 further opportunities in 37 additional hospitals, of which 5 are designated "special institutions." It has not been possible at this time to analyze these figures to determine the exact number of institutions in which systematized training can be provided, especially as regards the basic sciences.

APPRENTICESHIP SYSTEM

The apprenticeship system, with all its limitations, is a very valuable form of training. Much depends upon the preceptor, as well as the amount of clinical material available. *It should not be undertaken without basic training.* The apprenticeship system often involves a very limited variety of methods; too frequently, training is narrowed to the observation of the work of one or a limited number of individuals. It should be supplemented by visits to various large centers for broader observation. It would seem that extra-mural faculties could be organized to supplement intramural teaching. Much may be said for the value of extra-mural assistantships, either in general hospitals or with individuals, as a means of supplementing special institutional training, and as a medium for entrance into individual private practice.

BOARDS

Undoubtedly, the greatest influence in education for our specialties in recent years has been the outstanding, unselfish and unprejudiced labor of the special examining boards. These boards were originally instituted for the purpose of elevating standards of teaching and of passing upon the qualifications of eye, ear, nose and throat practitioners. They have now become a strong guiding force in determining educational trends, and rightly so. The attitude of these boards has never been too critical; it has always been constructive. For the present, at least, teaching can be appraised by no more effective method than by the results of examination. Analysis of cumulative data has revealed certain glaring deficiencies in instruction, especially in the basic sciences. It is a legitimate function of these boards to aid in the correction of weakness and to stimulate the interest of the graduate schools and special hospitals engaged in the training of prospective ophthalmologists and otolaryngologists. It is to the credit of the boards that proposed changes and correction of deficiencies in training have placed no restrictions upon educational freedom. They have advocated no rigid programs or curricula. Suggested changes, based on appraisal of their diplomates, have been favorably received by individuals and by institutions. Graduate schools and hospitals for training should eagerly seek the advice and aid of these boards in arranging programs and courses which will meet the minimum requirements for certification. The results of recent examinations of the output of certain hospitals have shown definite tangible evidence of improvement in the teaching of pre-clinical fundamentals very largely due to the requirements for certification.

It would require considerable labor, time and expense to scrutinize and evaluate teaching in each of the one hundred and thirteen institutions listed as offering training. It would be necessary to ascertain the extent of their equipment, the amount of available clinical material and how it is utilized, the library and laboratory facilities, and to determine how well their curricula are balanced. If such a survey could be made, it undoubtedly would have a very stimulating effect upon graduate medical teaching. One can recall the basis for rating "Class A" Undergradute School of Medicine. It might be possible to obtain funds from one of the great foundations, or from the A.M.A., or to have the expense pro-rated among the various interested special societies. Surveys could very well be supervised by the Advisory

Board, and include all of the medical specialties. One of the functions of the Advisory Board is the formulation of policies in education for the specialties. Incidentally, it is our good fortune to have able and experienced representatives from each of our boards upon the Committee on Graduate Education.

AFFILIATIONS

How can the criticism of unorganized, unsystematized teaching and training be met, and the weakness of special hospitals and institutions be corrected to provide a more completely rounded-out training for special practice? Stop to consider the excellent eye, ear, nose and throat hospitals, institutions and infirmaries we have, with abundant clinical opportunity in which teaching is not well organized, and in which little or no supervised instruction in basic subjects is, or can be, provided. Can the deficiencies in basic training be surmounted by the affiliation of special hospitals with university medical schools? Would the standards of future graduate education thus be raised? Is this a practical remedy for our greatest deficiency and weakness in graduate medical education?

We have witnessed the successful operation of such a plan in a few instances. A review of the location of our special hospitals reveals that in nearly every instance some such provision could be developed, with or without direct merger.

With the imperative demand for basic training, such union, affiliation, or even consolidation is necessary, practical, and would seem to be good economy. Increasing taxation may limit future private endowments and bequests. It will be unfortunate if some institutions refuse to recognize the difficulty of continuing on their present basis and the certain advantage of affiliation. Staff organizations may require revision; even broader interpretations of bequests may be necessary, but individual and institutional selfishness should never prevent the coalition of endowed institutions with desirable teaching centers, when ways and means can be found. Such mergers should be encouraged with due regard for the better training of specialists, as well as for the care of patients.

Improved teaching opportunity has never resulted in curtailment of care of patients. The training of better and more competent eye, ear, nose and throat practitioners is obviously in the interest of patients who are treated in special hospitals.

GRADUATE SCHOOLS

Financial aid is concentrated on undergraduate teaching, whereas graduate teaching is for the most part self-supporting. Aside from endowments, it has been estimated that less than one-half million dollars is actually spent on graduate medical teaching, and the larger part of this goes for administration. This sum, of course, is entirely inadequate. Graduate teaching is a heavy tax on the resources of an institution. Not many of our schools have been able to engage extensively in this work. More financial support must be provided before expansion in graduate supervision for advanced degrees can become universal.

Our universities are recognizing their great opportunity to direct and correlate graduate and post-graduate medical preparation and training in every special field of medicine. At present only about ten universities in the United States and Canada are prepared to include the basic sciences in graduate training for ophthalmology and/or otolaryngology. The few instances where graduate students are subsidized by foundations, universities or hospitals providing basic training are on the increase, but they are decidedly limited in number. May I be permitted to cite an example with which I am best acquainted? At the University of Minnesota four special eye, ear, nose and throat residencies in large municipal hospitals, ten or more fellowships at the Mayo Foundation, and four in the University Hospital now are under direction of the Medical Graduate School. None of these are in special eye, ear, nose and throat hospitals. These general hospitals are enabled to provide basic as well as clinical training, leading to a degree.

HIGHER DEGREES

The desirability of a higher medical degree as one of the credentials for certification reverts to the question of affiliation of hospitals with universities which can offer a degree. The attainment of a master's degree in ophthalmology or otolaryngology or both would be some assurance of proper basic training. It would constitute additional evidence of qualification to enter special practice. Its requirement would, for special practice, parallel the undergraduate requirement before entering the field of general practice. Would it not also aid in distinguishing the well-trained from the self-announced, partially- or poorly-trained specialist? Would possession of higher degrees or a certificate of

service in a recognized three year residency or fellowship not obviate necessity of formal examination by the boards? At present there is no legal qualification other than that established when the practitioner's training is questioned if he appears as an expert witness. One can visualize the possibility that state examining boards may, at some future time, require definite proof and certification of high ability to practice a specialty.

THESES AND RESEARCH

Many graduate students overlook or deny the advantage of possessing a higher degree. They distinctly object to the requirement of the thesis, or piece of "original research," usually demanded. Many of us have been sympathetic auditors to these objections. So-called "original" research by graduate students frequently involves unnecessary work, too often it is not undertaken in the proper spirit, and results in no appreciable contribution to medicine. Pure research should invariably be encouraged in the right type of individual. As a requirement for an advanced degree, or as a yardstick of fitness to practice, it frequently proves valueless. As a substitute for a thesis, a comprehensive review, with abstracts, of some subject adequately covered in the literature would prove equally valuable as a measure of the candidate's ability. The whole subject of higher degrees as well as of certificates of basic training and their influence upon standardization of organized teaching deserves serious consideration in this evolutionary period of advanced higher medical education.

BASIC TRAINING CENTERS

Another possibility suggests itself for solution of the problem of inadequate basic training. Could certain medical schools be designated as centers for intensive pre-clinical training, where the candidate will be required to spend three, four, or six months in *preliminary* advanced study and laboratory work? Thereby such subjects as special anatomy, embryology and histology, physiology and biochemistry, neurology, pathology and bacteriology could be taught to lay a foundation for further hospital clinical work. Didactic lecture courses in immunology and allergy, heredity, endocrinology, especially the relationship of general diseases to ophthalmology and otolaryngology could all be added. Such basic centers should be distributed in convenient geographical locations; for example, one each in Boston, New York,

Philadelphia and Baltimore in the East; in the Middle West and South, one in Chicago, Saint Louis, New Orleans and certain state universities, such as Michigan, Ohio, Iowa, Minnesota and others; in the West, in Denver, Portland, San Francisco and Los Angeles. It may be unwise to prophesy that this plan would be workable, or that students would profit by such a radical arrangement. One can easily appreciate its advantages in preventing educational duplication, if proper inter-institutional cooperation in the matter of basic and clinical training can be effected. When a higher medical degree is desired, credit should be guaranteed for all work accomplished in any approved center. Ways and means could doubtless be found by coordinated effort for bringing clinical institutions into close relationship with these basic centers, thus overcoming one of the greatest difficulties now being encountered. The suggestion is offered for consideration. As a matter of fact, it is not a very radical proposal.

TEACHING

Training involves teaching. Teaching is an art. Many good clinicians are not good teachers. Some of our best instructors write little and contribute infrequently to scientific gatherings. By reason of their vast experience and superior judgment they are able to supervise training of younger men, and to inspire and stimulate them. The good clinician teaches careful history taking, accuracy of observation, methods of arriving at a correct diagnosis, and application and technique of proper therapeutic measures; he also impresses the student with the collateral relationships to other branches of medicine, to previous disease, to allergic states, neurologic factors or heredity. The staff member who "gets by" with perfunctory work, or who holds a staff position for the prestige it affords is quickly appraised. His position should be filled by some younger man of ability and interest (who has been certificated), whose demands of private practice or interest in avocations do not preclude time for teaching. Younger men should be encouraged to teach and to concentrate in the field of their greatest interest.

To exemplify tact and diplomacy, to demonstrate candor without rudeness, to explain limitations of diagnosis or treatment without discouragement, all constitute a part of the teaching art. The greatest skill should be taught in the handling of patients who have already consulted a colleague, not to discredit the pro-

fessional standing or worth of the latter, either in the matter of diagnosis or treatment; it is invariably easy to make the patient feel that he has had excellent advice, and that any supplementary opinion or modification of treatment is given only in a spirit of helpfulness.

PUBLIC REGISTRY

Our graduate student is launched in practice, independently or as assistant to an older man. One of his first obligations is to undergo examination for certification; this should be impressed upon him throughout his training. In passing, I would like to remark that while we have lists of diplomates that we use in referring patients to distant communities, how can the public distinguish between the diplomate and the untrained man? Its frequent gullibility notwithstanding, the intelligent public, at least, desires to discriminate in its choice of ophthalmologists and otolaryngologists. Would a public registry of all certificated specialists be a very radical proposal?

AFTER CERTIFICATION

Finally, regarding post-graduate training. It is a subject by itself, but one in which each one of us is interested. Once away from metropolitan centers, where opportunities for clinical observation are numerous, it is sometimes difficult for those whose location is remote to follow the growth and expansion within our own special field. It is almost impossible under some circumstances to do all the reading and study desirable. The value of frequent attendance at meetings, of travel to clinical centers, occasional refresher courses or instructional courses such as are offered by this Academy, all maintain and stimulate intellectual alertness. The ideal scientist must be a student for life. He should endeavor to give as well as to accept. To paraphrase a quotation, "Nobody grows old by merely living a number of years; people grow old by deserting their ideals." "In the central place of the brain, there is a wireless station; so long as it receives knowledge and sends out information, so long are we young."

ADDRESS OF THE GUEST OF HONOR

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OPHTHALMOLOGY AND RESEARCH

Sir John Herbert Parsons, D.Sc.
F.R.C.S., F.R.S.

LONDON, ENGLAND

Before reading to you my address, I wish to express my very high appreciation of the honour that is being done me in inviting me as your guest. This is the fourth occasion on which I have been invited to America to address ophthalmological societies and each of those occasions is a red-letter day in the calendar of my life.

With regard to societies and meetings of this kind, I do not think that addresses such as I am about to inflict upon you, or even papers, are really of great advantage at such meetings. The great advantage is in the intercourse with men who are interested in and striving to advance studies which are common to those who belong to such societies. The interchange of ideas which occurs, and above all, the friendships which are formed, are perhaps the greatest possessions which anyone can attain.

It is a long way between England and America and the opportunities for intercourse between English ophthalmologists and Americans is comparatively limited. Doubtless, with the gradual abolition of space which is taking place, those facilities will be increased in the future. But, I must say that at the present time I have some sympathy with what must be the feelings of all of you, I think: that three thousand miles of sea between you and Europe in its present condition is not an unmitigated disadvantage.

* * *

The members of this Academy all belong to highly specialized branches of medicine. The unparalleled increase of scientific knowledge in modern times has inevitably led to the growth of

specialism in every department. The day of the Admirable Crichton has passed forever. It is futile to shut one's eyes to this fact, as is often done. On the contrary the position must be faced boldly, for specialism undoubtedly has its dangers, which must be studied and circumvented. It seems to me that the welfare of mankind in the immediate future depends more upon coordination of existing knowledge than upon the acquisition of new knowledge; and in many respects the former is the more difficult task — the latter is quite able to take care of itself, at any rate in the domain of the physical sciences.

Most of the papers and discussions which we hear at such meetings as this are of a highly specialized nature. An address such as that you have done me the honour to ask me to deliver is an opportunity for surveying a broader field, of attempting a synoptic view.

The reactions of every living animal, upon which its survival depends, are responses to the impacts of various forms of physical energy derived from its environment. In the lowest forms of life the response to any adequate stimulus is a crude tropism. Evolution to a higher plane in the animal scale is associated with, and indeed determined by, increased differentiation of the receptive organs and correspondingly increased differentiation of the resultant responses. In the higher animals the differentiation has led to the development of many receptors, far more numerous than the classical five senses of man. All these receptors are receptors of physical stimuli, whether derived from the outer world or from within the body. The receptors for mechanical stimuli are not confined to those of the tactile sense proper. In fishes the elaborate lateral line system responds to differences of pressure in the surrounding fluid medium, and hearing, both in fishes and higher animals, is but a further sublimation of response to rhythmical pressure changes in the surrounding medium. The proprioceptive receptors in the muscles, tendons and joints are but endogenous receptors of mechanical changes in tension of the parts. There are a host of chemical receptors — true chemical receptors in the mouth and on the body of fishes, almost atrophied to mere vestiges in man, further elaborated into taste and smell organs both in fishes and higher animals; for in both of these the actual stimulus is chemical. Many such occur in the organism itself, for hormones were first revealed as chemical stimuli, although their function does not end there. Perhaps the most remarkable of these are the chemical products of nervous

activity which themselves act as stimuli in the synapses and end organs in the form of histamine, acetylcholine, etc. The receptors or radiant energy subserve responses to change of temperature, but the most "glorified heat spots," to use Sherrington's dramatic term, are in the retina and subserve vision.

The simplest response of the animal to a physical stimulus above the level of a mere tropism is reflex action. Here the stimulus excites an end-organ, gives rise to an afferent nerve impulse which is transmitted through one or more intercalary cells to an effector cell, an efferent nerve and an effector organ. Chemical activity seems to dominate every step in the process. In some cases it is definitely the intermediary between the stimulus and the nerve impulse; perhaps the best example is the pupil reflex to light where the optic nerve impulse is started by a photochemical substance, visual purple or other such. Every nerve impulse in every nerve is invariably associated with an electrical response, which is probably the manifestation of ionic activities and therefore chemical in character. The discovery of adrenaline, histamine, acetylcholine, etc., indicates that every synapse and every neuro-effector junction is the seat of chemical activity.

The fundamentally important part played by reflexes in even the highest animal economy has been exhaustively studied by Sherrington and his pupils. They have shown how, as the result of summation, facilitation, inhibition, and so on, the most complex and apparently purposive responses are brought about. Indeed they have done much to bridge the gap between the activities of the lower levels of the nervous system and those higher levels which subserve conscious life.

At some point in this chain consciousness arises. Of consciousness we have no criterion but our own individual experience. In man reflex acts are unconscious acts, but higher activities at any rate show some analogy to reflex acts. The constellation of physical stimuli arouses perception of an experience suffused with meaning, which, in turn, arouses a conation towards the appropriate response. It is probable that lower in the phylogenetic scale the reflex act is associated with a crude undifferentiated sentiency.

We are accustomed to associate a particular sensation with the response of one of the higher so-called sense organs — touch with a tactile organ, hearing with the ear, vision with the eye, and so on. But just as a pure reflex is a physiological abstraction so is a simple sensation a psychological abstraction. It is

true that in a given experience one sensation may be so predominant as to mask the complication produced by the others. Indeed, one sensation may be predominant in the life history of an animal, and upon it its survival may appear to depend. Thus it is certain that in many animals, such as the dog, the sense of smell is prepotent, and there is little doubt that one of the most important factors, if not *the* most important factor, in the supremacy of the primates and man was the development of the prepotency of vision. None the less the meaning of an experiential presentation — to use psychological language — is derived from the presentation as a whole, and this is an integration of all the sensations derived from all the incumbent stimuli. This "pluri-receptive summation and interference" (Sherrington) by means of which the reactions of receptors of different modality are integrated is a biological mystery. There is indeed a similar integration on a higher level than this perceptual plane, for the presentation as a whole forms a pattern (Gestalt) which possesses meaning which cannot be explained by the mere algebraic summation of its parts.

From these introductory remarks it will be gathered that the study of normal vision demands no small knowledge of physics, chemistry, physiology, neurology, and psychology. As you all know, the study of clinical ophthalmology demands also a knowledge of general medicine and surgery, pathology and bacteriology.

The importance of physics is early impressed upon the student, for, at any rate in the early part of his career, the correction of errors of refraction will be his chief source of income. He must therefore know a good deal about optics, for nature has ordained that the end organ of vision shall be constructed on the principles of a photographic camera. The curious psychological fact must have struck most teachers that medical students are inherently averse from mathematics. My own experience is that they will show an intelligent interest in geometrical proofs, but that they jibe at an algebraical or trigonometrical expression. This is the more to be deplored in that opticians as a rule have had to submit to a much more intensive training in the theory of optics. I once expressed my regret to a distinguished physicist that many ophthalmic surgeons had a very elementary idea of the optics of retinoscopy. His reply was that one need not be a watchmaker in order to make good use of a watch. Admitting the cogency of this argument, the fact remains that the most efficient use of the ophthalmoscope, the slit lamp, corneal micro-

scope, etc., can only be attained by the thorough knowledge of the underlying optical principles. *Verbum sap.!*

The problem of visual acuity is primarily a purely physical problem and was first elucidated by astronomers. The discrimination of two points is simply the problem of the resolution of an optical instrument, and depends like it upon the formation of images by an approximately homocentric optical system with spherical and chromatic aberrations and diffraction at the margin of the pupil, and a receptive screen with a definite size of grain. But when all the physical conditions have been thoroughly established it is still found that the eye is a living organ and does not behave quite like a camera or a telescope. Though the physical conditions remain stationary the eye behaves differently in different circumstances. Thus it is found that there is a very much higher sensitivity to contour discrimination than to the resolution of two points of light. Whereas the former is of the order of a visual angle of at least 30 or 40 seconds of arc the latter is as low as 4 or 5 seconds of arc. This fact cannot be explained on purely physical grounds. The sensitivity of the receptive screen, the retina, and the mutual physiological inductive changes (contrast effects) which are set up in neighbouring areas of the retina result in a discriminative capacity which far exceeds that of any other sense organ in any animal. Fortunately for the physicists they discovered this fact empirically long ago and by means of the vernier applied it to their measuring instruments. Hence the extreme accuracy of physical measurements to which this form of discrimination can be applied, and their inaccuracy when it is not applicable, as in photometry. Physicists in their jibes at biological research often forget that their criteria are themselves based on biological data.

The presence of an intermediary chemical activity between the physical stimulus and the nerve impulse in vision may be regarded as proved by the discovery and investigation of visual purple. It is true that this is positively proved only for scotopic vision, i.e., vision under dark adaptation and weak light. It is almost certain that the much more complicated phenomena of photopic vision under light adaptation and greater intensities of light are also due to photochemical intermediaries which have not yet been isolated. The activities of adrenaline, pituitrine, and other hormones, and of histamine and acetylcholine suggest that there is a chemical intermediary in all cases of nerve excitation.

The researches of Keith Lucas, Adrian, A. V. Hill and others have educed some fundamental facts in the chemistry and physics of the impulse in the nerve. Every nerve impulse is accompanied by rhythmical changes in electrical potential, and these are of the same nature irrespective of the type of nerve — whether motor, sensory, proprioceptive, etc. The response is of the all-or-none type, i.e., a stimulus causes a maximum response or none at all, and there is a refractory state during which a stimulus of any strength is ineffective. Increase of intensity of the stimulus increases the frequency of the waves of response but does not affect their amplitude.

The electrical responses which accompany stimulation of the retina are much more complex, as might be expected, since a chain of several neurones is involved. The study of the electroretinogram of late years, especially by Granit and his colleagues, has thrown much light upon the highly complex functions of the retina. In typical form the curve shows a latent period, a small negative change of potential (*a*), a sharp rise of potential (*b*), a gradual fall followed by a slow positive wave (*c*), and a positive rise (*d*) when the stimulus is removed. There is little doubt that these complex waves are the integration of at least three waves, one negative and two positive.

The Purkinje experiment proved long ago that the visual impulse started in the neuroepithelial layer of the retina. It then passes through the bipolar cells to the ganglion cells whose processes end in the primary optic centres, whence another neurone carries the impulses through the optic radiations to the occipital cortex. It is attractive to regard the neuroepithelium as analogous to the epithelial end organ of a tactile nerve; the bipolar cell as a neurone of the first order analogous to the posterior root ganglion cell; the retinal ganglion cell as a neurone of the second order analogous to the mesial fillet; and so on. It must, however, be remembered that the retina and optic nerve are embryologically parts of the central nervous system, and there is now ample evidence to show that many of the functions of the retina can be best explained by bearing this fact in mind.

The evidence of the extraordinarily high discriminative capacity of the fovea shows that there is a strict dissociation of the impulses derived from neighbouring cones. The anatomical basis of this dissociation is found in the fact that each foveal cone has its own individual bipolar cell and ganglion cell — whereas in extramacular regions several rods and cones form synapses

with one bipolar cell, and several bipolars with one ganglion cell. At the same time there is evidence even in the macular region that summation of impulses can occur between neighbouring areas of excitation, and this is far more marked in peripheral regions of the retina. Thus it has been proved that such summation occurs by visual tests and also by the electrical responses in the retina and nerve. It was proved by Ricco and has been amply confirmed that the threshold stimulus in the macular area depends upon the quantity of light, i.e., the same quantity of light may be concentrated in a small spot or spread over a larger area. The anatomical basis for the summation is to be found in the amacrine and horizontal cells of the retina. So far as foveal vision is concerned these facts appear to be contradictory, and can only be reconciled by physiological explanations. The minute discrimination of foveal vision demands an inhibition of the summation which occurs in different circumstances. All the facts of spatial induction (simultaneous contrast) prove the reciprocal action of neighbouring areas of the retina upon each other when stimulated; and that this reciprocal action takes place in the retina and is not at any rate entirely dependent upon activities in the higher centres is proved by the effects of such stimulation upon the electrical responses. We owe to Granit the most exhaustive experimental proof of the facilitation, summation, inhibition, etc., of impulses in the retina just as they were proved by Sherrington and his pupils to occur in the spinal cord.

That these facts are not merely of academic interest is shown by the complex phenomena of vision experienced in every day life. It is only as the result of recent experiments that we are obtaining some idea of the causation of the evil effects of glare in interference with visual acuity and the production of discomfort, headache, and so on. By the careful experiments of Stiles, a physicist at the National Physical Laboratory, a numerical criterion of the effects of peripheral illumination on central vision has been obtained.

The persistence of vision as the result of momentary stimulation by light shows that the retina acts differently from a photographic film. The response to a single short stimulus is a series of waves of sensation, several of which may in favourable circumstances rise above the threshold and give rise to recurrent vision. Repeated short stimuli give rise to flicker which fuses into a continuous sensation if the repetitions occur sufficiently often. It has been found that the critical frequency at which

flicker disappears is dependent entirely upon the brightness of the resulting sensation and is independent of the wave-length of the light. This is the Ferry-Porter law which holds good for central vision, but when the critical frequency of flicker for peripheral regions of the retina is measured under different conditions of central and peripheral illumination complicated results are obtained, as has been shown by the admirable experiments of Lythgoe and Tansley. All these results show the importance of reciprocal activities of various parts of the retina; and some of them seem to indicate that the activities initiated in the cones may have an inhibitory effect upon those initiated in the rods and vice versa.

The visual phenomena to which we have already referred show that the eye must be an efficient optical instrument. That this may be so the media through which the light passes must be transparent, and the curvature of the cornea must be kept constant. In order that the latter requirement may be fulfilled the pressure of the fluid contents of the eye must be kept practically constant and above the atmospheric pressure, so that the walls of the globe may be kept in state of tension. The transparency of the media demands that the cornea and lens shall be free from blood vessels, and therefore some mode of nutrition independent of proximity of blood vessels shall be provided. Since the nutrition of the lens and vitreous depends upon the intraocular fluid, a stagnant fluid at constant pressure which would suffice to keep the tension normal will not suffice to keep these structures healthy. Movement of some kind or other must occur in this fluid.

The theory of the formation of the intraocular fluid which has held the field up to recent times is the filtration theory which is specially associated with the name of Leber. Clinical observation and experiments on animals seemed to prove that no process of true secretion occurs, and that the facts are adequately explained by the difference between the intracapillary and the intraocular pressures. It is not to be supposed that the earlier investigators ignored other possibilities, such as the importance of osmotic changes. For my own part, after reviewing all the evidence available at the time, I always taught my students that the facts appeared to be "not inconsistent with" the filtration theory. Evidence has accrued of recent times to show that osmotic interchange is more important than was once thought. Much of this evidence depends upon minute physical and chemical measurements which are perilously near the border line of

weighings carried to four places of decimals! At the same time it must be admitted that our knowledge of colloid chemistry has increased so much that the body of evidence in favour of the intraocular fluid being a simple dialysate is much stronger than it was. Perhaps one of the strongest arguments is the greater concentration of chlorides in the aqueous as compared with the blood plasma, for on the dialysation theory one would expect negatively charged ions to be driven through the capillary walls in order to maintain thermodynamic equilibrium with positively charged colloid ions (proteins) in the blood.

On the other hand, A. M. Walker, working in Professor A. N. Richards's laboratory at Philadelphia, has shown that the concentration of reducing substances in the aqueous humour of frogs is about 30 per cent less than that in plasma; and that of inorganic phosphates, 60 per cent less. In higher animals and man the deficiency of phosphates was uniformly present; that of reducing substances, though less marked and less constant, usually recognizable. The urea concentration in aqueous humour from rabbits, dogs and man is on the average 30 per cent less than in plasma. A similar result was found for uric acid. The author concluded that the choroidal and ciliary epithelium exhibit selective qualities not possessed by capillary endothelium or by glomerular membrane and that neither cerebrospinal fluid nor aqueous humour is formed by simple processes of filtration or dialysis.

The natural enthusiasm which recent results merit and have received tends to obscure the rôle which filtration plays in the process. For though these biophysical and biochemical reactions must now be admitted to be fundamental to the process of lymph formation they are completely explanatory — if that — of only static conditions. Under those conditions the intraocular pressure represents the resultant of the intracapillary pressure (say, 50 mm. Hg) minus the difference between the osmotic pressures of the aqueous and plasma (30 mm. Hg), i.e., 20 mm. Hg. But the intraocular pressure is never static. It follows passively normal changes in the blood-pressure due to pulse and respiration. It undergoes large rises due to activity of the extrinsic ocular muscles and the movements of the lids. Large changes in the general blood-pressure are accurately reflected in it except in so far as they are modified by local vasomotor and chemical effects. If, however, the general changes persist, as in cases of hyperpiesis, compensation occurs and the intraocular pressure resumes its

normal level. Convection currents are also set up in the anterior chamber owing to the difference in temperature between the iris and cornea.

Hence the atomic and molecular changes which have been proved to occur are associated with molar changes which alter the hydrostatic conditions. These are specially marked under abnormal conditions. If the aqueous is evacuated, e.g., by paracentesis, the capillaries dilate, their walls become more permeable and filtration of fluid takes place. The fluid thus formed more nearly resembles the blood-plasma in containing more protein than the normal aqueous. If again the eye is massaged the intraocular pressure falls. Seeing that this pressure is dependent upon experimental error. I am always rather suspicious of chemical the volume of the contents of the globe and the other conditions are not materially altered, the fall of pressure must be due to the mechanical expulsion of fluid from the eye. Cases of secondary glaucoma indicate the chief site of exit, for in them the angle of the anterior chamber is blocked. There can be no doubt that the rise in intraocular pressure which occurs in secondary glaucoma is due to blockage of this "filtration angle," whereby the aqueous is denied free access to the canal of Schlemm.

It has been shown that if the intraocular pressure is raised so as to exceed that of the venous pressure the veins collapse. This results in a reflex rise of arterial pressure so that the circulation is restored. The process may in some cases be repeated until the intraocular pressure is equal to the arterial pressure, and the circulation stops. The canal of Schlemm, however, is a venous channel in the substance of the cornea, and therefore does not collapse even when the intraocular pressure is moderately raised. Filtration can therefore occur through its inner wall — unless the latter is rendered impermeable by adherent iris, etc. — and an exit is thus provided for the excessive fluid and the normal intraocular pressure is restored. The meridional fibres of the ciliary muscle are inserted anteriorly into a scleral "spur." There is reason to think that when this muscle contracts on accommodation it pulls the spur backwards, thus tending to keep the canal of Schlemm open.

The permeability of the capillaries is increased in inflammatory conditions, e.g., iridocyclitis, and a plasmoid lymph, rich in protein, is formed. This causes a rise in intraocular pressure, and there can be little doubt that, since in these cases the filtration angle is not otherwise obstructed, and is indeed generally

abnormally open, the high tension is due to the difficulty of filtration of the large moleculed colloid proteins into the canal of Schlemm.

Many other facts might be adduced to show the importance of filtration in the maintenance of normal and the relief of abnormally high intraocular pressure. Neither dialysation nor filtration, nor both together, suffice to explain all the facts of normal and abnormal intraocular pressure. The dialysation theory has to fall back upon the variable permeability of the capillary walls in the various tissues of the body to explain the differences of lymph-pressure and constitution; and this variable permeability is at present only explicable on teleological grounds.

The long established explanation of the mechanism of secondary glaucoma depends upon the application of the filtration theory to the anatomical conditions found in these cases. Even the most ardent advocates of the osmotic theory of the formation of the intraocular fluid regard the so-called "filtration angle" of the anterior chamber with the canal of Schlemm as the principal site of exit of the fluid, and in general concur in the accepted view for secondary glaucoma. The very ingenious theory of Priestley Smith for the explanation of primary acute glaucoma also involves blockage of the exit channels. The constriction of the circumlental space in the hypermetropic eye by the continued growth of the lens is insufficient in itself to bring about the crisis, which is said finally to be due to venous congestion of the ciliary processes. Recent biochemical work tends to lend support to this exciting factor, for it has been shown by Dale and others that histamine-like bodies are found normally in many tissues of the body and are produced readily by antidromic impulses in nerves (cf., Lewis's "triple response" in the skin). These bodies produce dilatation of the capillaries, and are liberated by all forms of radiant energy. Duke-Elder has shown that the histamine-like response can be obtained in the albino rabbit's iris by stroking it, and that the more widespread axon-reflex is abolished by cocaine — a typical example of the triple response.

Priestley Smith's theory, however, does not explain the not infrequent cases of acute glaucoma in myopic eyes, and fails entirely to explain cases of chronic glaucoma. No one with wide experience of the latter disease can be satisfied with the tentative explanations which have been advanced. The treatment of such cases by trephining, etc., has been founded upon the theory of blockage of the filtration angle, and in spite of its crudity and

empirical nature is fortunately successful in preserving vision in a large proportion of cases. There are other cases in which it quite unexpectedly fails. There are, moreover, many cases in which increased intraocular pressure is never proved, and the presumption that it occurs is merely a slavish adhesion to current dogma. I have had rare cases of deep cupping in both discs, with full fields and normal visual acuity persisting for several years, yet eventually developing paracentral scotomata and finally constriction of the nasal fields. One must conclude that chronic glaucoma is not a single entity, but embraces cases in which the ætiological factors are different.

Recent biochemical researches on histamine and similar metabolites and on the nature of the vitreous suggest possibilities which only further research can prove or disprove. Specially interesting in this connection is the chronic primary glaucoma associated with epidemic dropsy met with in India. In the 1934-5 epidemic 1695 cases were treated in Calcutta (Kirwan). Although the disease is regarded as due to feeding on stored, par-boiled rice, it is quite different from beri-beri, and cannot be regarded as a deficiency disease. The interesting fact from the present point of view is that Kirwan found evidence of large amounts of histamine or at any rate an "H-substance" in the aqueous humour of a large proportion of the 23 cases he was able to examine. The albumin in the aqueous was also increased so that the ratio of albumin to globulin, which is normally 0.44 to 1.0, was in the ratio of 2.3 to 1.0, the crystalloids remaining normal.

I shall refrain from further discussion of these metabolites because I must confess that the independent variations in the calibre of the capillaries which have been proved by Krogh and others have introduced further complexity in estimating the hydrostatic conditions in various parts of the vascular system.

The work done on the vitreous has been almost as revolutionary as that done on the intraocular fluid. Like it, I do not think it is as revolutionary as appears at first sight. It may be considered as proved — at any rate for the sake of argument — that the vitreous is a gel and that the fluid which constitutes the sol is in every respect identical with the aqueous. The latter fact conforms with the filtration theory. It is stated that the greater concentration of micellæ at the periphery, etc., is in itself sufficient to account for the "structure" which has wrongly been described as a hyaloid membrane; and that similar concentrations and coagulations elsewhere account for slit-lamp and his-

tological "structures" which have been described in the interior of the vitreous. It is to be remembered, however, that the fœtal vitreous originates from definite structures derived from the retina and is full of blood vessels derived from the central artery of the retina and comprising the hyaloid artery and veins. It can scarcely be doubted that the fibres of the suspensory ligament are permanent relics of embryological structures and are not due simply to micellar aggregations. Normally the hyaloid vascular system disappears and the vitreous gel which replaces it is an inert non-vascular jelly nearly resembling the jelly-like material which is formed by the degeneration of the granulation tissue of a so-called Meibomian cyst.

So far as the non-ionised constituents and the albumin and globulin in the vitreous are concerned they are in identical proportion with those in the aqueous. The vitreous, however, also contains a mucoprotein and residual protein, and it is upon these that its behaviour as a gel depends. The investigations of Baurmann, Redslob, Duke-Elder, and others have shown that these proteins have two iso-electric points at about p_H 4 and p_H 9, and it is upon these that the turgescence and deturgescence of the vitreous gel depends. The maximum change of volume of fresh vitreous gel occurs at about p_H 8 to 9, and consequently we may expect that the volume of the vitreous is increased if the fluid contents are abnormally alkaline. Investigations of the alkalinity of the blood in chronic glaucoma have led to conflicting results, but it can scarcely be doubted that the considerable pressures exerted by a swollen vitreous may be an important factor in the pathogenesis of glaucoma.

The crystalline lens, like the vitreous, is a non-vascular organ, and like it is not susceptible to inflammation, so that the old terms hyalitis and phakitis imply ignorance and should be discarded. The chief pathological condition met with in the lens is cataract, and clinical observation over many years has led to the accumulation of a vast body of information as to the types of cataract and the concomitant conditions under which they occur. None the less our ignorance of the fundamental causation of cataract is abysmal. There can be little doubt that the solution of the problem depends upon the discovery of the normal metabolism of the lens and the factors which lead to its upset. Hence it is not surprising that a large amount of attention has been devoted to the biochemistry of the lens, and the remarkable advance which has been made in biochemical methods of recent

years has led to the revelation of many facts which cannot but be of fundamental importance in spite of the fact that they cannot at present be coordinated into a satisfactory theory.

The most important chemical constituents of the lens are the salts and proteins. Spectroscopic examination reveals many metals, of which the most important are sodium, potassium, and calcium. The potassium content diminishes with age, but the calcium is relatively constant (Adams). The normal lens contains more potassium than sodium, the reverse of the aqueous. It contains more potassium and phosphorus, less sodium and chloride, and the same amount of calcium as blood serum. The proteins consist of euglobulin, and α-, β-, and γ-crystallins. β-crystallin is soluble in water and decreases with age, a factor in the normal process of sclerosis of the nucleus of the lens. The crystallins are rich in tyrosine, cystein, and leucine — amino-acids which tend to form melanins on exposure to ultra-violet light, thus accounting for the normal and pathological pigmentation of the lens.

Owing to the absence of a blood supply the lens is dependent for its metabolism on an autoxidation system which has been proved to be identical with that which occurs in muscle (Hopkins). It is carried out by means of a reversible oxidation — reduction reaction, the catalytic agent of which is a cystein-like substance, probably identical with the glutathione found in muscle. This substance contains an SH group, which on oxidation changes into an SS group. Thermostable substances, of which the chief is probably β-crystallin, effect reduction of this product ($SH \rightleftharpoons SS$).

In senile cataract the potassium almost disappears, and the calcium may be eight times the amount of the normal (Adams). The total protein and β-crystallin are diminished. α-crystallin is most easily precipitated and is peculiarly sensitive to precipitation by calcium (Tsuji); its coagulation is probably the chief cause of the opacity in cataract. Glutathione diminishes and finally disappears. In senile cataract there is no significant change in blood-sugar, as compared with the increase which is concomitant with diabetic cataract, nor in the calcium content, as compared with its diminution in tetany and aparathyroid cataract.

Biochemically the essential factor in cataract is the coagulation of the proteins, and many important factors in this process have been discovered in recent years. In general, coagulation of proteins occurs in two stages; (*a*) denaturation, probably by hydrolysis, whereby the colloidal system becomes more labile;

(*b*) agglutination. Any form of radiant energy — heat, luminous, ultra-violet, radium — can cause coagulation. Ultra-violet rays alter the permeability of the lens capsule (Duke-Elder), diminish the efficiency of the autoxidation system (Adams), and render the proteins more vulnerable to variation in hydrogen ion concentration and salt concentration, e.g., calcium (Burge). Changes in the capsule cause alteration in osmotic pressure and hence in concentration of electrolytes. Deformation of the fibres leads to mechanical strains. Further, the lens proteins are organ specific, especially α- and β-crystallins, but investigations of their serological properties have proved contradictory. The relative parts played by these factors in the development of various types of cataract are obscure, and have not yet led to any satisfactory prophylactic or therapeutic results.

Apart from the experimental production of cataract in animals by various forms of radiant energy it is easily produced in rabbits by administration of naphthalene and other allied poisons; dinitrophenol, for example, used for slimming, has produced posterior cortical cataract in girls. The occurrence of cataract in tetany due to parathyroid deficiency when correlated with the excess of calcium in cataractous lenses suggests a definite association with the rôle of calcium in metabolism. Bourne and Adams found that the blood calcium was fundamentally unchanged by prolonged naphthalene dosing in rabbits. Yet they have shown conclusively that the blood calcium may play a determining effect of naphthalene on the eye; for an oats and cabbage diet, which prevents naphthalene cataract, causes a high blood calcium, and is associated with the deposition of crystals, probably calcium oxalate, in the retina; whereas a bran and carrots diet causes cataract and toxic symptoms in conjunction with naphthalene, and is associated with a low level of blood calcium. Recently Yudkin and Arnold have produced cataract in albino rabbits by administration of lactose or galactose, the latter being the more potent: this can be explained by disturbance of calcium metabolism.

I think I have given sufficient examples to show that laboratory experiments have materially advanced our knowledge of the physiology and pathology of the eye. Many other advances of a similar but more general nature doubtless have a distinct bearing on ophthalmological problems. Thus Tansley and others have proved conclusively that vitamine A is essential to the metabolism of visual purple. It is easy to produce night-blindness in rats by deprivation of this vitamine in their diet; and indeed

Charpentier, working with Granit, has used these animals for the purpose of analysing the electroretinogram. E. Mellanby's researches have shown that avitaminosis produces widespread changes in the nervous system, especially in the afferent nerves, and this may have a bearing on the ætiology of retrobulbar neuritis, whether as a manifestation of disseminated sclerosis or not, and possibly upon the ætiology of some lesions associated with the trigeminal nerve. It is by no means improbable that the metabolism of the eye structures is profoundly modified by disturbances in the internal secretions of glands, such as the thyroid, pituitary, adrenal, and the genital glands. Polyhormonic preparations have, indeed, been recommended as a cure for incipient cataract by Siegrist and others. Most competent observers have pronounced this method of treatment a complete failure. In spite, too, of the immense amount of work which has been done on immunology the practical results must be regarded as disappointing. *Parturiunt montes, nashetur ridiculus mus!* We may all agree that vaccine treatment is often beneficial, and sometimes curative, but there is a lamentable discrepancy between the frequency of its application and the successful results.

These advances in knowledge have by no means been neglected by clinicians, and praiseworthy attempts have been made to apply them to clinical problems. Great ingenuity and great perseverance have characterized these efforts. Only too often enthusiasm has outrun discretion, and the earlier reports of startling success have been followed by discouragement and ultimate admission of failure — or more commonly, silence. For my own part, I am convinced that the future advances of ophthalmology, and indeed of medicine in general, can be attained only by persistent pursuit of these more purely scientific efforts correlated with careful and equally truly scientific clinical observations.

For it must be carefully borne in mind that scientific research is a method and a mode of logical thought vitalized by intuition and imagination. Though many, perhaps most, of the outstanding discoveries of science have been made in the laboratory, scientific research is emphatically not the perquisite of the laboratory. If such were the case, astronomy could not be classed among the sciences in spite of the fact that it owes so much to laboratory discoveries. The physical processes which underlie most biological problems are so complex that few of these are solved by what I have elsewhere termed the "synthetic" method. The fact

that a cataract can be produced by the application of radiant energy does not solve the problem of cataract. We learn most about cataract by clinical observation and experimentation, i.e., by accurate correlation of the relationship of complex conditions. Just as psychological problems are rarely explicable upon a purely neurological theory, so biological problems are rarely solved by purely physical methods. Both modes of approach can be made truly scientific, and the scientist should admit that his scorn of empiricism is not wholly justified.

Every case which we are called upon to treat is a biological problem, and we are paid fees for the express purpose of investigating these problems. Whether we do so scientifically or not is a matter of method; and whether we make any great discovery or not is not even solely a matter of scientific training, but depends also upon inborn factors in the observer, such as imagination and intuition. For intuition, unscientific as the concept appears to be, surely plays a large part in scientific discovery. How did Gonin get the idea of sealing up the holes in a detached retina? If it was pure chance he is still to be credited with the discovery that only in those cases in which the holes were sealed up the retina became re-attached.

Discoveries have indeed been made as the result of action upon entirely false premises. Such was von Graefe's discovery that iridectomy affords a cure for acute glaucoma. He first performed the operation on the theory that iridectomy, often performed in those days for optical purposes, reduced the normal intraocular pressure — a wholly erroneous observation.

Our knowledge of the physiology of the normal intraocular pressure is still very scanty, and that of pathological increase of tension still more so, yet considerable success has been obtained in the treatment of chronic glaucoma by the many drainage operations which have been devised. If, as I think, they are all very crude methods of dealing with a little understood pathological condition, they at least have the pragmatic sanction that they are often successful in retaining sight. They remind me of Michael Foster's criticism of electrical stimulation of the brain, which he compared to playing the piano with a broomstick.

While we must all be profoundly dissatisfied with the utilitarian results of the application of biochemical and other forms of more "purely" scientific research, and also with the groping advances of hit-or-miss empiricism, we may yet rest assured that unmistakable progress has been made in our knowledge of oph-

thalmological problems and in our treatment of diseases of the eye. It is not given to everyone to be a biophysicist, a biochemist, a physiologist, or indeed, a pathologist. For such it is encouraging to know that he also can do useful research work; and it is still more important that all who have to deal with patients should be thoroughly trained in clinical methods, and that their clinical training should not be limited to their own specialty.

Recent numbers of the Archives of Neurology and Psychiatry have contained a valuable series of papers on the training of the neurologist and the psychiatrist. The arguments which apply to neurological training apply equally to ophthalmological training; and I cannot do better than quote the words of my colleague, Dr. F. M. R. Walshe: "So I believe," he says, "that while technical methods of investigation and experimental studies have now to be included in the essentials of a complete neurologic training, clinical observation remains the foundation of neurologic medicine, and on it all else must be built. Only the adequately trained clinical observer can evaluate the relationship of the results of laboratory and other technical methods of investigation to the symptoms of disease presented by the individual patient, for it is the individual that is the material of our study, be it remembered, and not an abstract thing called 'disease'."

The importance of the training and acumen of the observer is admirably expressed in the words of your great authority on medical education, A. Flexner. He writes, "There is a widespread impression that the scientific quality of medical education and practice is in some fashion dependent upon the part played by the laboratory. This is not the case. Science is essentially a matter of observation, inference, verification and generalization. Not only is the part played by the active senses the essential criterion of science; one may go further — the vast and complicated paraphernalia of science are merely means of extending their scope."

I have no hesitation in stating that in my opinion clinical medicine is better taught, and therefore better practised, in England and in the United States than in any other part of the world; and this is largely, if not chiefly, due to the fact that British and American students have not only freer access to the hospital patient than in other countries, but are compelled by careful notetaking themselves to investigate the signs and symptoms of disease and to exercise their own wits in arriving at a plausible diagnosis.

SYMPOSIUM ON MANIFESTATIONS OF DISEASES AFFECTING CEREBRAL NERVES SUPPLYING EYE, EAR, NOSE, AND THROAT DUE TO INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM

* * *

EYE INVOLVEMENT IN DISEASES OF THE CENTRAL NERVOUS SYSTEM*

DONALD J. LYLE, M.D.

CINCINNATI

WITH FEW exceptions, diseases of the central nervous system center about the base of the brain, an area of the greatest importance in ophthalmologic neurology. The blood borne meningitides, vascular lesions, encephalitis, the demyelinating diseases, cerebro-spinal syphilis and many of the degenerative changes, especially the juvenile forms, present as their most prominent and most constant signs, ocular nerve involvement. Frequently these symptoms are the initial manifestations of the presence of the disease.

At the base of the brain, in the middle fossa, lies the optic chiasm and the intracranial portion of the optic nerves surrounded by the chiasmal cistern containing cerebro-spinal fluid (Fig. 1). Under increased intracranial pressure this fluid is forced into

* The charts presented, with the exception of Chart I, are selections from a much larger series of cases representing the material from the review of over seven thousand necropsies covering about a ten-year period. This study was made at the Cincinnati General Hospital with the help of Dr. John McGavic. Only those cases were selected with fairly complete eye history. Then only those with "head permission" were retained as we were desirous of having brain reports. This work would have little value had it not been for the complete macroscopic and microscopic examination of the central nervous system by Dr. A. R. Vonderahe of the Department of Neuro-anatomy of the University of Cincinnati.

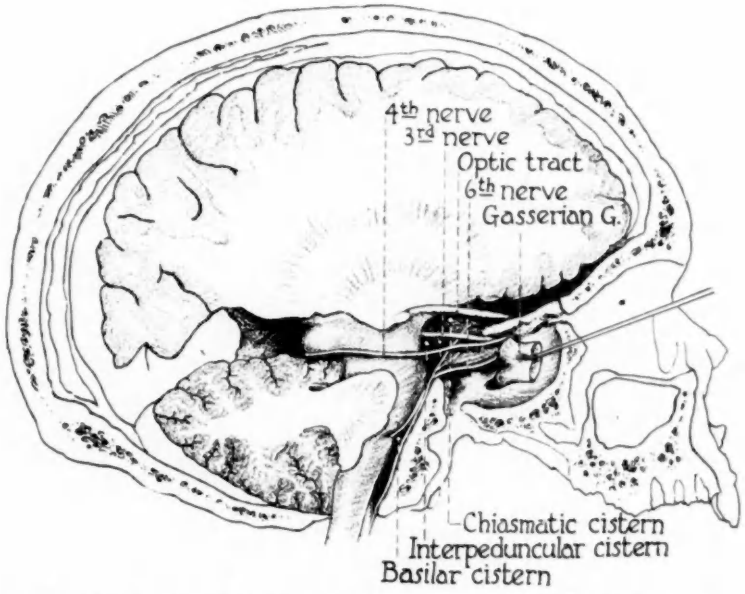

FIG. 1 — (Lyle) — The relative courses of the optic, oculomotor, trochlear, and abducens nerves in their passage through the subarachnoid cisterns from the brain to the orbit. Modification from Wolff's Anatomy.

the optic nerve sheaths, which are continuations of the meninges, to cause papilledema. Vascular lesions and inflammatory changes are frequent in this location resulting in neuritis and atrophy of the optic nerve. The motor nerves to the ocular muscles pass through this area on their way to the orbits, and may also become involved, producing ocular palsy.

The posterior fossa contains the cerebral peduncles of the midbrain which are separated by the interpeduncular space (cisterna interpedunculares) through which pass the oculomotor nerves. The trochlear nerves emerge posteriorly and sweep forward around the sides of the midbrain, through the lateral cisterns. The abducens nerves ascend through the basilar cistern along the anterior surface of the brain stem from below. These nerves converge as they pass forward to the orbits.

The basal cisterns are the chief locations for basal meningitis and meningo-vascular syphilis. The midbrain, which contains the nuclei of the oculomotor and trochlear nerves, is a site of election for many disseminated as well as local diseases (Fig. 2). It is here that lethargic encephalitis produces its chief lesions. Superior encephalitis of Wernicke is confined to this area.

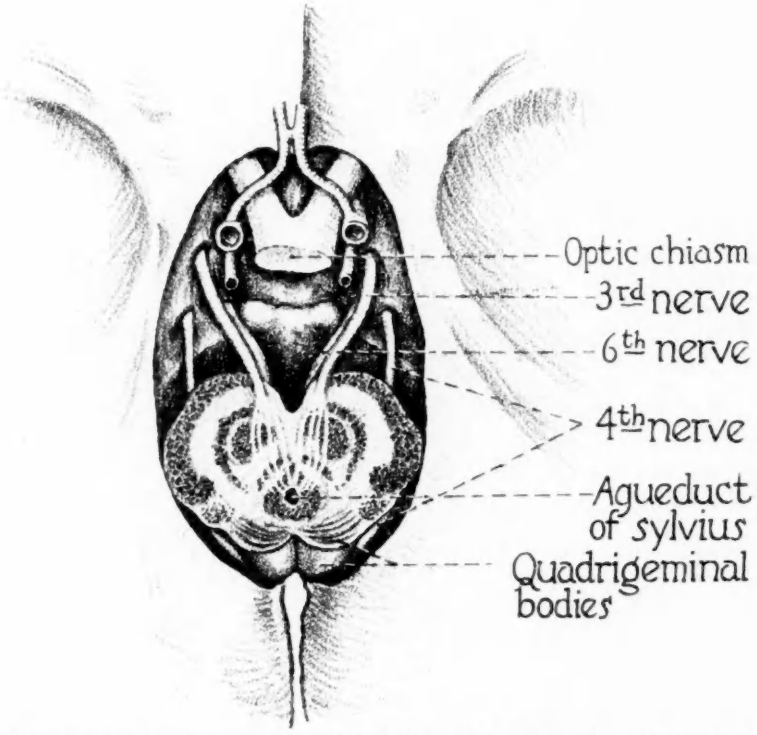

FIG. 2 — (Lyle) — A cross section through the midbrain at the base of the skull showing the oculomotor nuclei and emerging nerves, the trochlear, abducens and optic nerves, and the cistern spaces. Modification from Wolff's Anatomy.

This sketch of the anatomy will serve as an introduction to the vast subject I shall endeavor to outline briefly.

MENINGITIS

Meningitis, in various forms, often involves the base of the brain, especially the interpeduncular space. Ocular palsies are common because the nerves to the eye musculature pass through this area. Aside from the occasional facial, trigeminal and auditory nerve involvement, the other cranial nerves are rarely affected. Pupillary changes are frequent and their character is indicative of the stage of the disease. They may be roughly classified as early irregular, advanced sluggish, and terminal fixed and dilated pupils. Photophobia is a common symptom. Optic neuritis is frequent in meningitis. Occasionally a localized optochiasmatic arachnoiditis occurs with rapid onset of retrobulbar neuritis and,

at times, papilledema. Optic atrophy may follow. In spite of the almost constant high intracranial pressure, choked disc is seldom found. There are possibly three reasons for this. First, the illness may be so acute and fulminating that death intervenes before the development of papilledema; second, the spinal fluid may be so thick (purulent types), or the inflammatory process at the entrance of the subarachnoid space surrounding the optic nerves may be so marked, that extension of the spinal fluid into the optic nerve sheath is not possible; and third, frequent withdrawal of spinal fluid, and more especially, the therapeutic "through and through" irrigation of the spinal canal may keep the tension low enough to prevent papilledema (Chart I).

CHART I — *Frequency of Ocular Nerve Involvement in the Various Types of Meningitis in Children. A Comparison with Encephalitis.*

Type of Meningitis	No. of Cases	Optic Nerve E.	N.	A.*	Extrinsic Muscles	Nystagmus	Intrinsic Muscles	Pressure
Tuberculous	45	4	2	1	25	9	38	41
Epidemic Meningococcus	34	–	–	–	9	12	18	31
Influenza	12	–	2	1	3	1	6	11
Streptococcus	12	–	–	–	4	2	8	12
Staphylococcus	11	–	–	–	3	2	3	6
Pneumococcus	8	–	–	–	3	1	3	7
Syphilis	5	–	–	1	3	—	4	—
B. Coli	2	–	–	–	—	—	—	1
Serosa	3	1	–	–	1	—	1	1
TOTALS	132	5	4	3	51	27	80	110
Encephalitis	39	7	–	–	12	6	14	35

* Note: E = papilledema. N = optic neuritis. A = optic atrophy.

Tuberculous meningitis, the meningeal form of miliary tuberculosis, is found particularly in children. It is essentially a basal meningitis as are all the types of blood borne meningitides, the lesions occurring usually in the region of the circle of Willis, the interpeduncular space, fissures of Sylvius and optic chiasm. The oculomotor nerves are very apt to become affected resulting in intermittent or constant strabismus. Alternating miosis and

mydriasis is common. Nystagmus is seen occasionally. Optic neuritis and papilledema occur infrequently. Pallor of the discs and beginning atrophy are rarely encountered as the course of the illness is not usually long and is almost uniformly fatal.

Meningococcic or epidemic meningitis is quite apt to involve the basal meninges producing rather constant eye symptoms. Oculomotor involvement is seen in the majority with pupillary changes in about half of the cases. The trochlear and abducens nerves are occasionally affected. Nystagmus is rather frequent.

Influenzal meningitis, most common in small children, varies in frequency according to the epidemic. It is usually very severe. The eye findings are similar to those of meningococcic meningitis save that in influenzal meningitis nystagmus is less frequent and the optic nerve is involved more often.

Acute purulent cerebro-spinal leptomeningitis (purulent meningitis) caused by the staphylococcus, streptococcus and pneumococcus is frequently a complication of nasal sinus or mastoid infection due to extension of the process, involving the convexity rather than the base of the brain. For this reason, aside from the abducens nerve which is involved in Gradenigo's syndrome, the eye findings are not remarkable. Pupillary changes are noted but oculomotor and optic nerve involvement is uncommon. When sinus thrombosis and brain abscess complicate the condition, eye symptoms may appear, especially if the cavernous sinus is affected or brain abscess causes pressure symptoms.

Syphilitic meningitis usually begins in the interpeduncular space and around the optic chiasm. All of the nerves at the base may become involved but the optic, oculomotor and abducens suffer most.

In acute serous meningitis there may be some papilledema due to increased intracranial pressure. Transient ocular palsies have been noted. Meningismus arising most often from acute infectious diseases shows fleeting ocular palsies and transient visual disturbances.

Chronic leptomeningitis is usually found in those who have survived an acute attack. A continuation of the ocular symptoms in a permanent and aggravated form due to involvement of the nerves in adhesions is quite frequent. There may also be blockage of the cerebro-spinal circulation by residual inflammatory products resulting in internal, or localized external, hydrocephalus which may induce papilledema or neuritis and finally secondary optic atrophy (Chart II).

CHART II — *Cases of Meningitis with Choked Disc.*

TUBERCULOUS MENINGITIS

No.	Case	Eyes	Lesion	Brain	Ventricles	Cerebral Vessels	Spinal Pressure
1.	S.S. f. 36-49	Choked Discs	Chronic Leptomeningitis Tuberculous Exudate	Edema and Congestion	Marked Dilatation	Tubercles Along Vessels	Not Noted
2.	E.G. m. 25-83	Choked Discs Retinal Hemorrhage	Tuberculous Exud. about Optic Chiasm and Base	Edema and Congestion	Slightly Dilated	Tubercles Along Vessels	Increased
3.	C.D. m. 24-112	Choked Discs	Leptomeningitis Tuberculous Exudates	Edema and Congestion	Dilated	Tubercles Along Vessels	Increased
4.	L.F. m. 31-549	Choked Discs Venous Engorgement	Leptomeningitis Tuberculous Exudates	Edema and Congestion	Exudates Block 4th Dilating 3rd & Lats.	Not Noted	Increased
5.	M.M. f. 31-427	Choked Discs Retinal Hemorrhage	Leptomeningitis Basal Tuberculous Exudate	Edema and Congestion	Laterals Greatly Dilated	Tubercles Along Vessels	Increased

SYPHILITIC MENINGITIS

1.	O.J. m. 30-306	Choked Discs Ptosis & Anisocoria	Leptomeningitis Basal Waxy Thickening	Edema and Congestion	Appear Normal	No Note	Slight Increase
2.	S.C. m. 31-519	Choked Discs Venous Engorgement	Leptomeningitis Waxy Thickening	Edema and Anæmia	Appear Normal	No Note	Increased

PURULENT MENINGITIS

1.	A.N. m. 21-35	Choked Discs	Pus in Interpeduncular and Chiasmatic Cisterns*	Edema and Congestion	Distended with Pus	Greatly Congested	Increased
2.	C.H. f. 34-75	Choked Discs	Purulent Basal Meningitis	Edema and Congestion	Distended with Turbid Fluid	Greatly Increased	Increased

* Note: "When the brain is removed a large amount of greenish yellow exudate is observed at the base filling the interpeduncular space and passing forward along the optic nerves . . ." pathologist's report.

CHART III — *Choked Disc from Cerebral Hemorrhage.*

No.	Case	Eyes	Hemorrhage From	Hemorrhage Into	Brain	Spinal Pressure
1.	M.A. f. 33-441	Choked Discs Retinal Hemorrhage	Anterior Cerebral Septum Pellucidum	Subarachnoid (Base) Intraventricular	Edema and Softening	Greatly Incr. Bloody
2.	L.R. f. 32-214	Choked Discs Conjugate Deviation	Middle Cerebral	Subarachnoid	Edema and Congestion	Greatly Increased
3.	L.K. m. 32-532	Choked Discs Vessels Engorged	Middle Cerebral	Subarachnoid (Base)	Edema Marked	Greatly Increased
4.	M.J. f. 30-609	Choked Discs	Basilar	Subarachnoid	Edema and Congestion	No Record
5.	A.L. m. 31-98	Choked Discs Retinal Hemorrhage	No Macroscopic Site Found	Subarachnoid Intraventricular	Edema Marked	Greatly Increased
6.	G.C. f. 25-284	Choked Discs Conjugate Deviation	Middle Cerebral Basal Ganglia	Subarachnoid (Base) Intraventricular	Edema Marked	Increased
7.	R.C. m. 22-28	Choked Discs	Multiple Cerebral and Extra Dural	Subarachnoid Intraventricular	Congested	Increased
8.	A.W. f. 28-425	Choked Discs Retinal Hemorrhage	Multiple Frontal at Base	Subarachnoid (Base) Intraventricular	Edema and Congestion	Increased
9.	A.M. f. 29-148	Choked Discs Nystagmus	Frontal Lobe	Subarachnoid Intraventricular	Edema and Congestion	Increased
10.	A.S. f. 35-300	Choked Discs Retinal Hemorrhage	Internal Carotid	Subarachnoid	Edema and Congestion	Increased
11.	N.C. m. 27-140	Choked Discs Sclerotic Vessels	Circle of Willis Corpus Straitum	Subarachnoid Intraventricular	Edema	Increased Bloody
12.	A.W. f. 28-425	Choked Discs Retinal Hemorrhage	Frontal Lobe Sub Pial	Subarachnoid Intraventricular	Congestion Softening	Increased

VASCULAR LESIONS

Local ocular symptoms depend upon the area affected by the vessel in which the vascular lesion occurs. Immediately following profuse hemorrhage occurring at the base of the brain, low grade choked disc may be seen due to increase in intracranial pressure (Chart III). There may be associated retinal hemorrhages caused by compression of the veins in the optic nerve. If the pressure be great enough to affect the ocular nerves, palsies appear, and if the high pressure persists, optic atrophy may result. Fixed dilated pupils, usually of uneven size, are more frequent in basal hemorrhages than in thrombosis alone. In embolism, pupillary changes are still less frequent.

Cerebral hemorrhages may be caused by a weakening of the vascular walls usually by arteriosclerosis or syphilitic endarteritis, frequently with hypertension. Hemorrhages most frequently affecting the nerves to the eyes are from the internal carotid, the anterior and middle cerebral, and the anterior and posterior communicating arteries.

A rather common occurrence in hemorrhage of the brain is conjugate deviation of the eyes (Chart IV). This condition is about twice as frequent in hemorrhage as in thrombosis. Besides lesions of the midbrain and pons, involvement of the eye motor center in the posterior third of the middle frontal convolution or along the course of the cortico-bulbar tract above the brain stem, especially in the knee of the internal capsule, may cause conjugate deviation of the eyes and often the head as well. The angular gyrus, when affected, is also productive of conjugate eye movement. Lesions along the efferent visual tracts, the corticofugal optic radiations to the eye muscle nuclei, apparently produce slow conjugate reflex movements. Association tracts between the visual sensory and related areas in the occipital cortex and the eye motor area in the frontal lobe, though of more or less speculative nature at present, appear to produce conjugate deviation of the eyes when affected. Chart IV shows a series of cases of conjugate deviation of the eyes with necropsy findings in the central nervous system.

Aneurysms, besides rupturing, may by distension cause transient attacks of visual or motor eye disturbances if the lesion is in a vessel in proximity to an ocular nerve. Some cases of migraine with or without ophthalmoplegia result from such dilatation.

Some forms of angiomata are of interest to the ophthalmologist. Angioma venosum by its size may produce increased intra-

CHART IV — *Conjugate Deviation of the Eyes*

No.	Case		Dev.	Location of Lesion	Nature of Lesion
1.	S.E.	33-443	L	Fronto-Temporal Lobe, L.	Hemorrhage
2.	W.S.	33-151	R	Fronto-Temporal, R.	Fracture-Hemorrhage
3.	D.W.	32-655	R	Parieto-Occipital, L.	Softening
4.	L.R.	32-214	L	Middle Cerebral, L.	Hemorrhage
5.	F.B.	32-543	R	Temporal Lobe, L.	Tumor
6.	J.A.	34-83	R	General Cortex	Softening-Atrophy
7.	M.S.	34-123	L	General	H'm'rhagic Encephalitis
8.	J.M.	34-391	R	Internal Capsule, R.	Hemorrhage
9.	J.L.	34-391	R	Parieto-Occipital, R.	Hemorrhage
10.	G.C.	25-284	L	Middle Cerebral, L.	Hemorrhage
11.	M.C.	28-424	L	Temporal Lobe, L.	Hemorrhage-Softening
12.	L.H.	26-371	R	General	Purulent Meningitis
13.	U.N.	26-402	L	Parietal, R.	Fracture-Hemorrhage
14.	M.P.	26-404	R	Parietal, L.	Hemorrhage
15.	R.A.	30-303	R	Fronto-Parietal, R.	Hemorrhage
16.	L.W.	29-394	R	Internal Capsule, L.	Hemorrhage
17.	E.D.	29-486	R	General	Hemorrhage-Edema
18.	H.H.	29-555	R	Midbrain	Hemorrhage-Softening
19.	R.C.	30-65	L	Internal Capsule, R.	Hemorrhage-Softening
20.	M.J.	29-55	R	Internal Capsule, R.	Softening
21.	F.S.	29-97	R	Fronto-Internal Caps., R..	Hemorrhage
22.	M.H.	26-208	R	Parieto-Occipital, L.	Hemorrhage
23.	U.N.	26-78	R	Occipital Lobe, R.	Hemorrhage-Edema
24.	M.S.	29-599	L	Internal Capsule, L.	Softening-Atrophy
25.	P.A.	36-200	R	Internal Capsule, R.	Hemorrhage
26.	M.G.	36-57	R	Fronto-Temporal, R.	Hemorrhage-Softening
27.	G.M.	36-25	R	Leptomeningitis, Extensive	Exudative Edema

R = right. L = left.

cranial pressure resulting in occasional choked disc. Arterial angioma (arteriovenous aneurysm) when adjacent to the orbit may cause exophthalmos from interference with the blood supply to the eye and orbit. In these conditions papilledema is fairly common.

Sinus thrombosis produces, in practically all instances, a congestion and dilatation of the superficial veins that drain into the involved sinus. The surrounding tissue is often edematous and discolored. In cavernous sinus thrombosis, though anastomosis with the anterior facial vein may lessen the severity, the retinal veins and those of the eyelids and upper part of the face and nose are dilated and there is edema of those parts with proptosis of the eyeball and progressive ophthalmoplegia. The trigeminal nerve is frequently affected. Occasionally papilledema, and retinal hemorrhages due to disturbances in retinal circulation, are present. At times it is difficult to differentiate these cases from those of leptomeningitis with extension into the orbit.

THE ENCEPHALOPATHIES

Encephalitis may be divided into:

A. Toxic type (due to intoxication), proliferative in nature.

B. Infectious type, infiltrative in nature.

Encephalitis of the toxic type shows proliferation in the vessels and pia-arachnoid membranes especially marked at the base of the brain, about the optic chiasm, temporal lobe and cerebellum. The midbrain is only slightly affected. On the other hand the infectious inflammatory type always, but not exclusively, involves the midbrain.

Typical examples of the toxic type are found in malignant hypertension, glomerulo-nephritis, toxemia of pregnancy and lead poisoning. In these cases there is usually an optic-neuro-retinitis with edema of disc and retina. Retinal exudates and hemorrhages are common. Where there is an increased intracranial pressure, papilledema is frequently present. These are the wet brains (so-called uremic), increased in weight with flattened convolutions and narrowed sulci. Visual hallucinations are occasionally noted. Visual fields may be constricted. Central scotomata are not common in the cardio-vascular-renal types, but are frequent in those due to organic poisons because of retrobulbar neuritis.

CHART V

Papilledema in Arterio and Arteriolar Nephro-Sclerosis

No.	Case	Eyes	Kidneys	Heart	Brain	Spinal Pressure
1.	W.J. m. 25-12	Papilledema A-S Retinitis	Vascular Nephro-Sclerosis Chr.	Hypertrophy	Edema	Increased
2.	L.G. f. 29-76	Papilledema Exudates	Vascular Nephro-Sclerosis	Myocardial Degeneration	Soften-ing	Greatly Increased
3.	H.L. m. 27-349	Papilledema A-S Retinitis	Vascular Nephro-Sclerosis Adv.	Dilated and Hypertrophy	Slight Edema	Increased
4.	H.K. f. 30-218	Papilledema A-S Retinitis	Vascular Nephro-Sclerosis Adv.	Endocarditis Myocarditis	Edema	Increased
5.	M.J. m. 29-55	Papilledema A-S Retinitis	Vascular Nephro-Sclerosis Adv.	Myocardial Fibrosis	Soften-ing	Greatly Increased

Papilledema in Glomerulo Nephritis

No.	Case	Eyes	Kidneys	Heart	Brain	Spinal Pressure
1.	B.V. f. 28-517	Papilledema Hemorrhages	Glomerulo Nephritis	Endocarditis Myocarditis	Edema	Increased
2.	I.M. f. 33-339	Papilledema Detachment Hemorrhages	Glomerulo Nephritis	Myocarditis Hypertrophy	Edema	Increased
3.	J.W. m. 32-505	Papilledema Hemorrhages Exudates	Glomerulo Nephritis (Vasc. Scler.)	Hypertrophy	Edema	Slight Increase
4.	A.D. m. 32-268	Papilledema Hemorrhages	Glomerulo Nephritis	Myocarditis Dilatation	Edema	Slight Increase
5.	B.S. f. 30-236	Papilledema Hemorrhages Exudates	Glomerulo Nephritis (Vasc. Scler.)	Pericarditis Hypertrophy	Edema	Increased

The cranial nerves are also more frequently affected in the latter types.

Chart V shows a series of cases of papilledema in hypertension and renal cases with necropsy reports of the principal organs involved. In the one series the hypertension picture predominates; in the other series there is chiefly and primarily a renal condition.

Infectious encephalitis, as in the case of toxic encephalitis and meningitis, involves usually most severely the base of the brain, especially the midbrain containing the oculomotor nuclei and their emergent nerves. The acute forms rather frequently show ptosis, strabismus and nystagmus. Facial paralysis occurs. Occasionally choked disc is seen. Special mention must be made of acute hemorrhagic superior polio-encephalitis of Wernicke which is limited to the area of the midbrain about the aqueduct of Sylvius, and therefore involves the nuclei and emergent oculomotor and trochlear nerves, resulting in internal and external (partial if nuclear) ophthalmoplegia. Encephalitis secondary to other intracranial infections may present ocular involvement if the inflammatory area includes the nuclei or nerves to the eyes.

Epidemic encephalitis (lethargic encephalitis) shows a predilection for the basal ganglia, red nucleus, oculomotor nuclei, substantia nigra and occasionally the tegmentum of the pons and medulla. In most instances the initial symptoms are diplopia and impairment of pupillary reflexes and accommodation. In the acute stage the extraocular muscles are nearly always involved. Optic neuritis is found occasionally, but choked disc is rare. Transient amaurosis and optic atrophy are very rare though spinal pressure is increased. Where lethargy is a prominent symptom, eye findings are frequent.

The sequel to epidemic encephalitis includes pupillary abnormalities. These may be irregularity, inequality, miosis or mydriasis. The pupils are often fixed, occasionally showing a true Argyll Robertson syndrome. One case of Argyll Robertson pupil was noted in a child who also had strabismus (Children's Hospital No. 7358).

Not infrequently, and more or less characteristically, oculogyric crises occur. In this condition muscles are thrown into spasm resulting in conjugate movements of the eyes, usually upwards. The crises may last from minutes to days. Or there may be paralysis, especially of vertical gaze, when the eyes cannot

be elevated. The lesion causing these symptoms is probably in the hypothalamus or its vicinity. Paralysis of lateral gaze frequently occurs. Nystagmus is not uncommon.

THE DEMYELINATING DISEASES

There is a class of diseases having a predilection for the myelin of the central nervous system especially the optic nerve, chiasm and tracts. The myelin degeneration is probably caused by neurotropic viruses usually associated with those producing the dermatropic manifestations of the acute exanthematous diseases. The chief characteristic of this class is early retrobulbar neuritis frequently without ophthalmological findings, especially in Schilder's type which usually begins in the occipital lobe. With other forms, optic neuritis and papilledema are occasionally found.

(1) Multiple sclerosis (disseminated sclerosis) in its primary form is usually a disease of adolescence or young adults. Next to cerebro-spinal syphilis and encephalitis, it is the most disseminating of all cerebral lesions. The white matter is involved in this condition, the optic nerve, chiasm and tracts being especially affected. By far the majority of all retrobulbar neuritis is due to multiple sclerosis. It is an early, and not infrequently, the first symptom. Occasionally optic neuritis and papilledema are found. Central scotomata are most frequent in this condition. Temporal pallor of the discs is common and often appears early. The intensity of this pallor apparently bears no relation to the visual function. The patient may see well with what appears to be a very advanced atrophy or may see poorly with a negative ophthalmoscopic papilla, the lesion being far back. Blindness is rarely complete. Striking remissions may occur. Early, often fleeting, ocular palsies are seen. Pupil changes are not infrequent. Nystagmus (nystagmoid movement), another characteristic symptom, though often a late one, is usually horizontal and is accentuated by fixation.

(2) Acute disseminated encephalomyelitis shows less spotty demyelination than that of multiple sclerosis. The oculomotor, trigeminal and facial nerves are involved in frequency next to the optic nerves, chiasm, tracts and higher visual centers. Ophthalmo-encephalomyelitis or encephalomyelitis optica (German) or neuropticomyelitis (French) consists of a subgroup of disseminated encephalomyelitis with constant visual involvement which is usually bilateral.

(3) Neuromyelitis optica (Devic) is another type with bilateral involvement of the optic nerves. Retrobulbar neuritis is the rule with occasional optic neuritis and papilledema.

(4) Diffuse peraxial encephalitis (Schilder) is probably another type of demyelinating disease which occurs in the young, possibly with familial tendency. Demyelination usually begins in the occipital and temporal lobes and extends forward so that cerebral blindness is present before the optic nerve is involved.

CEREBRO-SPINAL SYPHILIS

Cerebro-spinal syphilis is the most disseminated of all diseases of the central nervous system so that a composite picture of encephalitis, meningitis and vascular involvement of all parts of the brain and its coverings is often present. In meningo-vascular syphilis the chief seat of the trouble is at the base of the brain. Probably over half of all ocular palsies are due to syphilis. These are generally peripheral in nature with ptosis, strabismus and loss of pupillary and ciliary action. Dissociated oculomotor nuclear palsies are less frequent. A fixed, rather than an Argyll Robertson pupil, is characteristic of meningo-vascular syphilis. The pupils are frequently irregular and unequal. The oculomotor is the most frequent of all the cranial nerves to become involved. Nystagmus is occasionally found. The trigeminal and facial nerves are also affected occasionally. Increased pressure is the rule so that choked disc is sometimes seen, probably most frequently due to localized external hydrocephalus. The fields show concentric constriction from involvement of the external fibers of the optic nerve. Secondary optic atrophy may result from these lesions. In congenital meningo-vascular syphilis optic atrophy appears to be commoner. Gummata are found on the dura and cortex but especially in the interpeduncular space of the midbrain necessarily involving the emergent oculomotor nerves. Another favorite site is the optic chiasm with resultant visual disturbances. Syphilomata give rise to symptoms both local and general similar to brain tumors. Involvement of the midbrain resulting in an Argyll Robertson pupil and paralysis agitans has been referred to as the syndrome of "mesencephalitis luetica."

General paresis is a meningo-vascular encephalitis resulting in general atrophy of all parts of the brain including the brain stem nuclei with incomplete or dissociated ocular paralysis, fleeting or constant. The pupils may be unequal, miotic, mydriatic;

most often fixed or showing an Argyll Robertson reaction which occurs in frequency only second to tabes dorsalis.

Tabes dorsalis has a predilection for the cranial nerves, especially the optic and oculomotor. The optic nerve, of all of the cranial nerves, is most often affected, undergoing an atrophy that is usually regarded as primary. Optic atrophy may occur very early in the disease and it may be the only symptom for a long period. Atrophy may become arrested, but usually goes on to blindness. The fibers in the temporal part of the disc are the last to be destroyed, producing the more or less characteristic concentric constriction of the visual field.

The next most frequent cranial nerve to be affected is the oculomotor which results in fleeting or permanent paralysis of incomplete or dissociated nuclear type. These palsies may occur quite early. The Argyll Robertson pupil is a most frequent and characteristic finding. The pupil may be miotic, irregular and unequal. Fixed pupils do not usually occur in tabes. However, in the late stages when optic atrophy is complete and the eyes are blind the pupils may be dilated and fixed.

DEGENERATIVE CONDITIONS OF THE CENTRAL NERVOUS SYSTEM

Amaurotic family idiocy (Tay-Sachs' disease) is a progressive condition of unknown origin resulting in ganglion cell degeneration of the central nervous system and retina. Vision is defective, the optic disc showing atrophy. There is a cherry-red spot in the macular area surrounded by a white halo. A juvenile form closely resembling the infantile type just described shows retinal pigmentation but no macular changes. Nieman-Pick's disease, found in infancy, resembles Tay-Sachs' in that it occasionally has a similar fundus picture (Chart VI).

Diffuse peraxial encephalitis of Schilder is mentioned again in this group as it may be due to softening in the white matter of the brain with destruction of myelin of degenerative nature. The condition usually begins in the occipital lobe. Cerebral blindness with a normal disc is a most frequent finding. A few cases of optic neuritis and choked disc have been reported.

Congenital extracortical axial aplasia of Pelizæus-Merzbacher is a sclerosis chiefly affecting the white matter, causing, among other symptoms, nystagmus and optic atrophy.

Hepato-lenticular degeneration, an atrophy of the brain, especially basal ganglia, frequently, with cirrhosis of the liver includes

CHART VI — *Degenerative Conditions of the Central Nervous System*

Name	Age - Character	Ocular Symptoms
Amaurotic Idiocy Tay-Sachs' Type Nieman-Pick's Type	Infantile 4-6 mo. Juvenile 5-6 yrs. Familial-Congenital	Cherry-red Macula Optic Atrophy Retinal Pigment
Diffuse Periaxial Encephalitis Schilder	Children and Young Adults	Cerebral Blindness
Congenital Extra- Cortical Axial Aplasia Pelizæus-Merzbacher	Young 1-5 yrs. Familial	Optic Atrophy Nystagmus
Pseudosclerosis Westphal-Strumpell Progressive Lenticular Degeneration-Wilson	Young Familial Adolescent and Young Adults	Corneal Pigment Ring Retinal Spots Optic Atrophy
Primary Optic Atrophy Leber	Puberty-Early Adult Familial-Hereditary	Optic Atrophy Central Scotoma
Friedreich's Ataxia Marie's Ataxia	Puberty-Early Adult Familial-Hereditary	Nystagmus-Palsies Optic Atrophy
Syringomyelia Syringobulbia	2nd to 3rd Decade	Horner's Syndrome Nystagmus-Palsies
Chronic Progressive Ophthalmoplegia Graefe	3rd Decade	Motor Ocular Paralyses Excluding Smooth Muscle
Myasthenia Gravis	3rd Decade	Ocular Fatigue & Palsies

the pseudo-sclerosis of Westphal and the lenticular degeneration of Wilson. A syndrome of corneal pigment ring, tremor and slow speech is characteristic. Retinal and nerve changes occasionally occur.

Hereditary primary optic atrophy of Leber is a slowly progressive condition which may become arrested or even regressive. There is a central scotoma with normal peripheral fields. The discs gradually assume pallor.

Friedreich's ataxia is due in part to a degeneration of the spino-cerebellar tracts with nystagmus as the chief eye symptom. Optic atrophy occurs occasionally. Marie's cerebellar heredo-ataxia is probably a variant of Friedreich's. In addition to nystagmus this condition shows ocular palsies.

Syringomyelia (syringobulbia) is a degeneration of the central glial tissues of the cord and brain stem. Horner's syndrome is caused by involvement of the sympathetic tract. Hydrocephalus, due to blockage of the foramina, with papilledema has been reported. The nuclei of the ocular nerves are not infrequently affected. Chronic progressive ophthalmoplegia of Graefe is a degeneration mainly in the floor of the third ventricle and the aqueduct of Sylvius causing progressive external, and later internal, ophthalmoplegia.

Myasthenia gravis, though it is really a myopathic and not a neuropathic condition, is mentioned because of its bulbar-like symptoms. There is a predilection for those muscles in most constant use. The first symptom is often ptosis. The muscles of the eyes tire readily and recover, at first, after rest (asthenic ophthalmoplegia). Later permanent paralysis appears with lagophthalmos.

NEURITIS

Neuritis, for want of time, will not be discussed in its entirety. It has been mentioned in connection with other intracranial diseases. Diphtheria, tetanus, lead and alcohol and other organic and inorganic poisons and toxins affect the ocular nerves producing paralysis and loss of vision.

There is a rare condition called interstitial hypertrophic neuritis (Déjerine-Sottas) caused by hypertrophy of the peripheral nerves and roots and sclerosis of the posterior columns which produces ocular symptoms. Miosis, fixed sluggish pupils, ocular palsies and nystagmus have been reported.

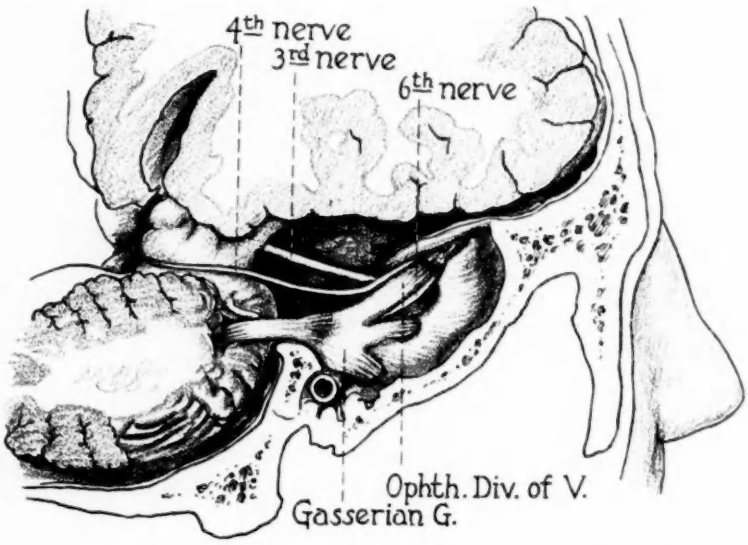

FIG. 3 — (Lyle) — The relation of the oculomotor, abducens and trochlear nerves to the Gasserian ganglion and especially to the ophthalmic division of the trigeminal nerve. After Wolff's Anatomy.

Herpes zoster ophthalmicus is an infection of the Gasserian ganglion principally. The nerves to the ocular muscles are in such close proximity to this ganglion that they are frequently, though not so reported, involved in the inflammation and swelling (Fig. 3). Paralysis lasts for weeks, after the acute attack has subsided, occasionally remaining permanently.

* * *

When invited to take part in this symposium, I was asked to try to show the great importance of the vast subject rather than present a detailed discussion. Because of limited time it was necessary to omit several important subjects. As the original purpose was a symposium for the discussion of "non-surgical diseases" I decided to adhere to the first intention and omit brain tumors, brain abscesses, and brain injuries because they are usually considered surgical cases.

The importance of diseases of the central nervous system in ophthalmology cannot be over-estimated. Just as the eye dates its development from an out-pouching of the brain with which

it remains associated in order to function, so the ophthalmologist must date his proper knowledge of the eye from the study of neurology from which his specialty cannot be divorced.

* * *

CHART I. A consecutive series of meningitis and encephalitis cases at the Children's Hospital.

CHART II. Diagrammatic reports of three types of meningitis with choked disc. A complete necropsy was had in each case. Note that the tuberculous and purulent meningitis cases had an internal hydrocephalus. The tuberculous meningitis brains had a yellowish exudate with tubercles which extended along the blood vessels. The syphilitic brains showed a paraffin-like thickening and exudate. The purulent forms showed brains bathed in pus which also filled the ventricles. Edema and congestion of the brain with increased intracranial pressure was constant in all cases.

CHART III. A report of a series of twelve choked discs from cerebral hemorrhage. A complete necropsy was had in each case.

CHART IV. Conjugate deviation of the eyes resulting from cerebral hemorrhage and other causes. This consecutive series indicates the frequency of hemorrhage as a caustive factor in this type of deviation.

CHART V. These charts are selections of typical cases from a much larger series. They show the similarity of involvement of the eye and vital organs of the body in malignant hypertension and glomerulo nephritis. A complete necropsy was had in every case.

CHART VI. The chief degenerative conditions of the central nervous system affecting the eye are usually found in early life. The senile and pre-senile degenerations do not have constant or frequent eye findings. Myasthenia gravis is included because, though it is a myopathic condition, it closely simulates nervous degeneration.

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In addition to the mass of clinical histories and pathological reports, which number nearly eight thousand, from which most of the material for this paper was gleaned, the following texts have been referred to. In some of the rarer conditions I have made free to summarize the general opinions of those who are able to write with authority.

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SYMPOSIUM ON MANIFESTATIONS OF DISEASES AFFECTING CEREBRAL NERVES SUPPLYING EYE, EAR, NOSE, AND THROAT DUE TO INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM

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COMPARATIVELY few otolaryngologists and ophthalmologists have been especially interested in the phases of neurology which they have encountered. Those who have *become* interested focused their attention on neurology involving acute infections necessitating surgical procedures. There remains a less spectacular, but none the less important, study represented by the title of our symposium. Unlimited possibilities for a combined neurological, ophthalmological and otolaryngological research, promising solutions to many of our disturbing problems may be foreseen.

We have heard the plea for closer cooperation so frequently, I hesitate to mention it again. The plea is trite, but it is also very necessary. Little has been accomplished in clarifying many central nervous system diseases with manifestations in our specialty. These diseases have been rather vaguely classed as degenerative or vascular in origin. The various syndromata are the result of disease which affects the pons, medulla, midbrain and cranial nerves. Many other neurological diseases, of which multiple sclerosis is an example, also produce symptoms discernible by the observing head specialist.

Recently, an article by Foster Kennedy called attention to one of the possible causative factors in some of these diseases. His very rational treatise dealt with the allergic factor. The theory impressed the reader with its logic. When we recall the symptoms encountered of known allergic origin and note the almost daily additions to our knowledge of the subject, it would seem impera-

tive that more specialists should join forces in attempting to solve our mutually perplexing problems.

A list of the more common symptoms encountered might be started with headache. Each of you can readily recall a patient who complained of this distressing symptom. The patient had undoubtedly gone from doctor to doctor without obtaining relief. The head specialist had sent her to a neurologist, who sent her to the allergist, who in his turn sent the distraught patient to an internist. The internist probably had her eyes and sinuses re-examined. It may be true that our discouraged patient found relief by having her feet manipulated. Nevertheless, her complaint was real to her and an unsolved problem to us.

Let us consider some of the other symptoms which combined effort may help to relieve, but which at present sometimes defy our best efforts. Dizziness, perhaps, is second only to headache in the frequency with which it occurs. Like headache, it occurs in many forms and is the result of many disorders. Dizziness may be described by the patient as a giddy sensation, a sensation of falling, of turning around themselves or rotation of external objects. They are unable to keep their balance, being thrown violently from one side to the other. They have a floating sensation. Things appear black before their eyes and they experience the not too uncommon justified drunken feeling. We are well acquainted with Ménière attacks and if we are able to produce relief it is usually a case of cut and try before we determine whether these attacks have their origin in the gastro-intestinal tract, the fore brain, cerebellum, the basal ganglia or the end organ of the vestibular apparatus. I have found the statement, that all dizziness is the result of vestibular stimulation, difficult to believe and more difficult to prove. Compensation in stability following loss of both vestibular end organs is so nearly complete, it seems improbable that dizziness as a symptom of vestibular disturbance could not also come from the same disordered mechanism which normally compensates for loss of vestibular function.

The lateral-pulsion of a patient, who has multiple sclerosis, may throw him involuntarily from his chair, or perhaps into the street in front of an approaching automobile. Death ascribed to suicide may follow the accident. The patient who staggers down the street evoking the knowing smiles of the passers-by may be suffering from cerebellar disease with its accompanying dizziness and incoordination.

Aphonia may attack similarly the singer who has spent many nights brooding about the overwhelming importance of her début, and the doughboy, green from a normal fear of the impending zero hour, or the patient having a paralysis of the vocal cords as the result of a destructive nuclear lesion. The differential diagnosis may be difficult, but the laryngologist is the one called upon to prescribe rest on the one hand or perhaps sign the death certificate on the other.

Proper elaboration of the various speech disturbances would require volumes rather than paragraphs. A few significant statements may stimulate the desire for a more complete understanding. Aphasia is a complete disorganization of the central mechanism for expression; while aphonia results from a local disturbance in the end organ of speech or larynx. The various types of aphasias indicate which part of the central system is diseased and therefore responsible for a loss or distortion of function. The congenitally deaf patient has the ability to use his larynx, lips, tongue and other end organs necessary for speech, but since he has never heard the sounds, of which speech is composed, the central mechanism is not equipped to produce articulate speech. Sounds can be produced, but the arrangement of sounds into the orderly variation recognized as coherent speech is lacking. When the accessory structures employed to form words are involved the patient has what is popularly known as an impediment in his speech or dysarthria. The patient may have a destruction of the centers which construct the pattern of speech perception. He is then able to speak but has no comprehension of the spoken word. His motor apparatus may be involved. In which case he is able to understand what is said but lacks the ability to reply. Our responsibility to a patient really is limited to the differentiation between the symptoms of aphonia and dysarthria as the result of local disturbances on the one hand and aphasia and dysphasia as a central manifestation on the other. This is a rough division with evident fallacies, but it is an attempt to clarify what, to me, has been a confusing group of symptoms.

Paralysis of the face is a most alarming symptom. Picture the emotional reaction of an individual whose mirror records the distortion of his features. These patients commonly consult the neurologist. In not a few instances, however, they have heard that facial paralysis is associated with mastoiditis and then they consult the otologist. A simple differential point aids the otologist

in the proper treatment of these patients. A facial paralysis of central origin is characterized by the preservation of function in the superior branch of the facial nerve. Function is retained because of the double innervation arising from each cerebral hemisphere which is distributed to the musculature supplied by the superior branch. A peripheral lesion involves all three branches. A simple test is also useful to determine whether function will return to the patient having Bell's palsy. If the face responds to Faradic stimulation, the function will probably return. If the response to Faradism is lost, the chances of complete recovery are slight. This last test originated from the unpublished observation of Doctor Tickle on facial paralysis. The significance of this important observation needs no elaboration.

Regurgitation of fluid or food through the nose may be the result of a benign condition such as temporary paralysis of the soft palate following adenoidectomy. The condition is easily rectified by properly conducted muscle exercises devised to stimulate movement. Paralysis of the soft palate may, however, indicate a bulbar involvement with a fatal prognosis. The differential diagnosis again is the important feature and usually it becomes the responsibility of the laryngologist.

Anosmia as a symptom is often lightly regarded by those of our specialty and probably also underestimated in importance by others. A recent work by Elsberg accentuates the importance of disturbances and loss of the sense of smell as indicating lesions of the central nervous system. His statements should impress us with the importance of thoroughly testing the function of the first cranial nerve.

Sudden loss of the sense of smell is immediately noticed by the patient. When a head cold precedes this loss, the anosmia is usually transient, and thus quickly forgotten. The more protracted nasal diseases of the ozena type gradually dull the sense of smell until it finally disappears. The location of causative pathology, therefore, may be difficult. A detailed history combined with thorough examination will usually be effective in making the diagnosis. The alert examiner is aware, at least, of the graver possibilities.

Involvement of the second, third, fourth and sixth nerves cause disturbances in vision, ocular palsies and distorted muscular function. These symptoms cause the patient to seek aid from the ophthalmologist.

Pains over the distribution of the fifth nerve frequently are interpreted by the patient as a certain sign of sinus trouble. Since the public has become so sinus-minded, it is not uncommon for a patient to make his own diagnosis and, almost with the same breath, dictate the treatment. It is difficult to give such self-determined patients the benefit of a complete unbiased examination, although the very situation makes the thorough evaluation of the symptoms all the more important.

Motor disturbances are much more frequently noted by examining physicians than either sensory or autonomic. The reason is probably the predominance of the objective element. It is none the less important to determine the sensory and autonomic changes.

Deafness is a symptom. It is a symptom of some underlying pathology involving not alone the end organ of hearing, but also the complex central and peripheral nervous system. How casually we admit the deafened patients to our office, and then, after a brief and cursory examination, relegate them to the "hopeless" category. It is true that a history of sudden unilateral deafness may cause the possibility of an angle tumor to flit through our minds, but we have slight comprehension of, or thought for, the many diseases of the central nervous system resulting in deafness. Realizing the frequency with which other cranial nerves are involved, it is rather surprising that the thought of deafness as a symptom of central nervous disease is slighted. The vestibular branch of the eighth nerve is subjected to frequent and thorough investigation, sometimes overestimating the importance of our findings. In considering the auditory division of the eighth nerve, however, we are so apt to think only of its terminal importance. Deafness, the most common complaint of patients visiting an otologist's office, is still perhaps the least understood of all symptoms. Progress has been made by studying the pathology underlying deafness. The work on the spiral ganglia done by Crowe and Guild at Johns Hopkins and the monumental work fostered by the Scientific Committee on Otosclerosis of the American Otological Society are two examples of the advance in scientific research which is progressing favorably. All earnest, honest and tireless workers would do well to utilize the knowledge of the neuro-pathologist and physiologist, as well as the rather overworked physicist, thus correlating the work to better advantage.

By presenting symptoms which so deeply affect the emotions of patients, I hope to cause a reaction on the part of some ear,

nose and throat specialist to the extent of further study, thereby clarifying his cloudy diagnostic acumen.

Perhaps it is unfortunate, but it is nevertheless true, that many patients seen by us have symptoms indicative of serious neurological diseases. These patients first seek our aid because the symptoms are manifest in our realm. We are apt to dismiss them briefly because of a lack of appreciation of their grave condition; or else carelessly overlook the obvious valuable data which is most important if early treatment is to be instituted. These patients wander about in a sort of medical no-man's land because of a hiatus in the reciprocal relations of the specialties. The average neurologist possesses insufficient knowledge of the head specialties and his lack of knowledge is probably only surpassed by the lack of neurological knowledge among those practicing ophthalmology and otolaryngology.

For many years I have had the privilege of an association with the members of the Neurological Department of the Post Graduate Hospital. This association gave me a good insight into the viewpoint of the neurologist. Many times, patients from the neurological clinic have been referred to the Ophthalmological and Otolaryngological Departments for opinions regarding the patients' status. The reports sent back to the neurologists were sometimes useless and frequently inadequate. I might add that this is at the same time self-criticism. The deficiency in these reports was due to an insufficient comprehension of the neurological problem involved. There has been a perfectly natural sequence to the situation. Neurologists have learned to do their own examinations of eyegrounds, fields of vision, hearing tests and vestibular test. In fact, neurologists competently test the functions of all cranial nerves. Of course, these tests may properly be delegated to the field of neurology, but it is surely obvious that these same tests are a most important part of our own routine.

In order to produce the best results it would seem desirable for us to broaden our scope to the extent of understanding the simpler principles of neurology with which we are confronted.

Those of us who have spent some time studying phases of neurology have, perhaps, posed before our confrères as knowing more than facts warranted. This pose may have been impressive to other members of our own specialty, but the effect on neurologists was a reaction of partially justified intolerance. This intolerance was perhaps not warranted in view of the op-

posite situation when the neurologist ventures an authoritative opinion on the fundus of the eye as opposed to the trained ophthalmologist.

In order to have a better mutual understanding, let us admit some of our own limitations. For example, we have placed too much importance on the deductions drawn from the effects of labyrinthine stimulation following the use of rotating chairs. The nuances in response and their interpretation, based on the stimulation of a mechanism producing such a complexity of reactions, seems, to me, subject to inaccuracies. One need only recall the many reflexes described so classically by Magnus and De Kleign to realize the complexity of effects resulting from the stimulation of that most intricate mechanism. The substantiated information derived from the rotation test in clinical practice is indeed small. The caloric test is perhaps more satisfactory, in that its stimulation is localized and its reactions slightly less complex. This test also is distinctly limited as to the definite information derived. Our tests for auditory function have recently been subjected to critical intensive studies which promise at least a few changes in our beliefs of many years' standing.

We can not be over-modest if we wish to avoid the chagrin which follows the destruction of our most elaborately drawn diagnoses when they are subjected to the illuminating verities of the necropsy.

In order to gain information relative to lesions involving the eighth nerve, it is necessary to again remind ourselves that our present information is inadequate. We are prone to consider the end organ of the eighth nerve as the location of all equilibratory and acoustic disturbances. Let us ask ourselves how accurately we are able to locate the causes of impaired hearing. How much do we know about tinnitus? Are we able in even a few cases to say that dizziness is without doubt a result of a disturbance in the vestibular apparatus? Is a patient's inability to walk straight due to central or peripheral pathology? Is the nausea and vomiting, which is supposed to be the result of vestibular disturbance, not in reality central? What tests have we at our disposal to prove our answers to these questions? The fact that very few of our lauded tests give definite information will be obvious. Even the tests upon which we have depended year after year are undergoing a readjustment.

Permit me to cite a few examples of failure in popular and widely accepted diagnostic combinations. A doctor's wife com-

plained of occipital headaches, dizziness, nausea, difficulty in locomotion, impaired hearing, and tinnitus in the left ear. Hearing tests verified the complaint of unilateral deafness. There was no reaction on the left ear to caloric test. No response was elicited from the vertical canal of the opposite side. My diagnosis was acoustic neuroma. The patient at present is progressing nicely in the second year following my examination. She has no symptoms except the impaired hearing. I recognize the fact that a lesion may still be present, but my private opinion is that I was originally wrong.

I will cite an interesting observation in this case. In doing the caloric test on the right ear, there was no vestibular response. When the patient's head was tilted back the nystagmus indicated activity of the horizontal canal. When the patient's head was returned to the upright position, the nystagmus continued. A refinement in technique is suggested whereby the testing of the horizontal or vertical canal may be done after an intervening period of rest.

I have also observed the absence of response from both vertical canals while both horizontal canals were active.

Absence of past pointing with normal nystagmus present following vestibular stimulation is not an uncommon observation. We might decide that this situation indicates the need of neurosurgery, but our decision would probably be modified if we had to accept the responsibility of operating.

I have placed a great deal of confidence in the differential test between labyrinthine falling and cerebellar falling as brought out in a modification of the Rhomberg test. It has been accepted as a fact that patients having vestibular involvement fall in the direction of the slow component of nystagmus. The direction of the fall is changed by altering the position of the head. The patient having a cerebellar lesion will fall in any direction regardless of the nystagmus or the position of the head. The direction of the fall usually being to the side of the lesion.

About two years ago I made a diagnosis of cerebellar abscess principally on this point. A radical mastoid operation entirely cured this patient. The possibility of a lesion still being present is recognized, but, I believe, no pathology remains.

The information we obtain from our hearing test is likewise limited. Glibly we recite the combinations of symptoms, such as loss of high tones, decreased bone conduction, positive Rinné, Weber referred to the good ear, and on these combinations place

a label of perceptive mechanism lesion. Now, of course, this may be and probably is correct, but just how much scientific proof can we furnish to substantiate our claim? Also, of how much value is an opinion that localizes a lesion anywhere between the end organ of the ear and the many cortical ramifications of cerebral neurons?

It is an interesting fact that the evidence of diagnosed clinical otosclerosis is infrequently found at autopsy. Also, it is a fact that extensive pathological lesions characteristic of otosclerosis have been demonstrated in preparations from patients who had no symptoms of otosclerosis during life.

In what respect do these indictments relate to our subject? Otologists are asked by the neurologists to test the function of the eighth nerve. To what degree are we able to furnish concrete information? If we test the seventh nerve, we can distinguish between a central and a peripheral lesion. The ophthalmologist can give a fairly accurate location of trouble in his field. In numerous instances we have gone so far astray as to state that as a result of our tests, the neuro-surgeon may search as we direct and there he will find trouble. In *diseases* of the brain there will be no surgery to check our flights of imagination. If we now have the temerity to direct neuro-surgery, is it not possible that our imagination will run riot in non-surgical cases? Our unleashed emotions are due to be curbed if our background lacks a thorough knowledge of neuro-anatomy and neuro-pathology. I dare say none of us are sufficiently venturesome to acquire that knowledge. Let us rest on our rightful laurels, knowing that the limited information we are able to furnish the neurologist, if correct, is valuable. We should acquaint ourselves with the function of the cranial nerves and the methods of testing their function. When we observe abnormalities in function not explained by conditions with which we are familiar, our next procedure should be a cross check with the neurologist in order to establish a diagnosis.

The cranial nerves which include autonomic innervation are the oculomotor, or third; the fifth, trigeminal; the seventh, or facial; ninth, or glosso-pharyngeal; and vagus, or tenth. We should also bear in mind the nerves of special sense, which may not possess autonomic fibres as such, but which have the power to produce violent sympathetic reactions following stimulation. The eighth nerve is, of course, a conspicuous example.

Much remains to be learned concerning the function of autonomic fibres associated with the cranial nerves. If recent literature is any indication, perhaps we are beginning to realize the major rôle which this system plays in many obscure diseases affecting the eyes, ears, noses, and throats of our patients. Since lesions of the central nervous system cause motor and sensory nerve disorders, it is obvious that the autonomic system must also be involved. The cause of some of the degenerative central nervous system diseases has been ascribed to the disordered vaso-motor system. We are familiar with the peripheral vaso-motor diseases as represented by vaso-motor rhinitis, angio-neurotic œdema, croup and labyrinthine disturbance. These phenomena illustrate the more startling phases of peripheral vaso-motor changes. There remain the less spectacular conditions evidenced by the atrophic or degenerative diseases.

The following quotation may serve to illustrate the effect of a vascular insult to the autonomic system. It is from Wechsler's *Clinical Neurology*. Speaking of acute bulbar palsy, he says: "This is generally caused by an acute vascular accident of which hemorrhage and thrombosis are the most common." Surely, we know of so-called "strokes" due to cerebral vascular insult without actual hemorrhage. Is it not also tenable that the vascular mechanism would operate to produce similar changes in the plexiform blood vessels around the bulb? Again to continue the quotation relative to the symptoms: "Usually the onset is sudden, with or without dizziness. The latter may, however, be severe enough to cause falling and be accompanied by vomiting. If the hemorrhage is very large there is a convulsion and death rapidly ensues. Generally, however, either there is no deep coma or consciousness is not lost at all, and the characteristic signs of bulbar palsy become acutely manifest. Occasionally they take a few days to develop. The common symptoms are dysarthria and dysphagia. The speech is nasal and the food regurgitates through the nose. Occasionally there is complete aphonia from involvement of both vocal cords. Whether the nucleus or the nerve be affected, the lesion is of the lower motor neuron, therefore, there is flaccid paralysis of the palate, pharynx, larynx and tongue, occasionally, also, of the muscles of the lips and face. Salivation is not uncommon, while vaso-motor disturbances of the face and a homolateral Horner's syndrome occasionally follow involvement of the medullary sympathetic fibres. These findings, combined with respiratory and cardiac embarrassment, alternating hemiplegias

and crossed sensory disturbances, contribute toward making the diagnosis." Evidence of autonomic involvement is ample and the vascular cause is evident. It still remains to determine the effects of the less violent vascular disturbances.

Bulbar compression with resultant sympathetic symptoms are not unfamiliar to those of us who have seen the result of cerebro-spinal fluid pressure imbalance following the spinal tap. In those patients I have observed one feature which remains indelibly impressed on my memory. The respiratory failure combined with a lack of cyanosis. The face, on the contrary, has the ashen gray appearance of profound shock. The heart, strangely enough, may continue to function long after the respiration has ceased.

I have tried a procedure which was effective in one case. The patient was under the care of Dr. Fred Graef and he proposed an exploration for cerebellar abscess. While the patient was under anesthesia and the operating room prepared, a spinal tap was done. A few drops of cerebro-spinal fluid was withdrawn, when the patient ceased breathing. The usual stimulants failed to restore respiration. I asked permission of Dr. Graef to try injecting air into the spinal canal. The rationale of this procedure being the equilization of pressure between the fluid of the posterior fossa and the spinal canal. In this case the method was immediately effective. Respiration was restored for twenty-four hours when, presumably, a second abscess, not found at the time of the operation, ruptured into the fourth ventricle.

The second time I tried the same procedure was on a patient already *in extremis*. She was breathing with great difficulty. Respiration was improved following an injection, but the patient died.

A few years ago, angina pectoris was named with finality as a coronary thrombosis, yet sympathectomy has in some cases furnished a reprieve. What this procedure may promise for the future in treatment of the sympathetic system of the head would seem in the realm of the ridiculous even to conjecture. I mention this procedure in spite of my objection to severing important nervous structures. It is uncomfortably similar to cutting the wires from a fire alarm box.

Dandy has produced clinical relief from the disturbing symptoms usually associated with the Ménière syndrome by selective section of the eighth nerve. It seems to me that the procedure should only be used as a stop gap until some intensive investi-

gation is done which will determine the underlying disturbance in physiology and the pathology. The fact that selective sectioning of the eighth nerve can produce relief from such symptoms forms a nucleus for further development along more conservative lines. In this paper I have purposely avoided discussing those syndromata which give evidence of involvement of the central nervous system and the cranial nerve simultaneously. This important phase of our subject will be covered authoritatively by Dr. Riley.

My awe of the neurological diseases was in no small degree caused by a vocabulary which to the uninitiated is awe-inspiring. However, it is not too difficult to comprehend the portion of neurological expression necessary to decipher their meaning. I have been an anxious observer on many occasions, helplessly standing by while the eminent consulting neurologist gravely conducted his most intricate examination. Then after summing up the situation in a language which was commonplace to him but incomprehensible to me proceed to extract from me a dumb nod of acquiescence. Friendly association with your neurologist will broaden your outlook enjoyably and the association, I assure you, will be for mutual enlightenment.

I would like to state a question which has been asked of me. "Why bother about diseases of the central nervous system affecting the cranial nerves?" "Why not leave that to the neurologist?" My answer will be my conclusion.

Most of us are old enough to have known intimately or by reputation the pioneers in our specialty. They were pioneers who developed the surgical part of our specialty to a perfection that needed little modification. The surgical procedures have been so standardized that anyone with surgical ability can perform them creditably. Do not blind yourselves to the fact that general surgery is including the more common operative procedures in all specialties. A specialist must be one who perfects himself above the average ability in his chosen field. Special new operative techniques which are superior to known and used procedures are becoming fewer in number. Pioneering in surgery is, therefore, limited in scope. This is the first point.

When the specialties first started few general practitioners or surgeons possessed the small amount of special training necessary to produce the best results. Therefore, they sent their patients to specialists. Through post graduate teaching, special subjects are becoming more generally understood. The general physician and surgeon with the broader training is giving his

patients adequate personal care. He only calls the specialist in more unusual cases, thus limiting the existing field for the specialist. This is the second point.

The specialist has had a training which unfortunately causes him to see the patient in terms of his own narrow domain. With the justifiable enthusiasm, he has forged ahead along his narrow path without enough regard for collateral highways and the intervening fertile fields. This lack of broader development has left him with a narrowed perspective. He is not sufficiently versed in the recent advances of medicine to apply the new knowledge in his own specialty. His usefulness to the profession as a whole is being limited. This is the third point.

We, therefore, have a physician whose dramatic advance was heralded by innovations in surgery and who now faces limitations. His work is being absorbed by medicine as a whole and the ignorance of the recent developments in general medical knowledge has curtailed his usefulness for a greater service to patients.

An example of such patients would be the topic of today's symposium. They arrive at the doorstep of the head specialist and through non-recognition go away unbenefited.

We speak of disease of the central nervous system with cranial nerve involvement. What a field for our mutual advance and what a benefit to our patients! It entails a study of the autonomic nervous system and many other fascinating subjects. My hope is that the papers of today's symposium will stimulate those progressive souls among us to again pioneer and enhance their value as specialists by developing the fertile areas lying between the main highways representing their several specialties.

SYMPOSIUM ON MANIFESTATIONS OF DISEASES AFFECTING CEREBRAL NERVES SUPPLYING EYE, EAR, NOSE, AND THROAT DUE TO INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM

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BY INVITATION

NEW YORK

A SYMPOSIUM before the American Academy of Ophthalmology and Otolaryngology offers an unrivalled opportunity for the presentation of material jointly of interest to the neurologist and the oto-ophthalmologist. I appreciate the privilege of being a participant.

Much of the diagnostic material absolutely indispensable to the neurologist is directly within the field of the oto-ophthalmologist. The neurologist, however, must be aware of the functional capacities of the structures of the head and neck and be able to recognize disturbances and disabilities connected with them; contrawise the oto-ophthalmologist. It is not of so much importance for the neurologist to be able to identify pathological conditions in the organs themselves supplied by the cerebral nerves, but it is of absolute importance to differentiate between normal and abnormal function in the organs of special sense and the structures of the head and neck. It is necessary, likewise, to determine whether the abnormalities observed are the result of implications of the nervous system, either peripheral, central or due to some local cause.

The title of this symposium includes only the central nervous system. The peripheral nervous system was not included within its scope. The various disturbances affecting the peripheral nerves are not so complicated, nor so numerous, as those which may implicate the origins, terminations and connections of the cerebral nerves within the central nervous system. Many of the factors

involving the peripheral course of the cerebral nerves are contributed by regional disease, injury or degenerative change, and, therefore, are not only more familiar but also perhaps more readily accessible and, therefore, easier to recognize.

The factor complicating the interpretation of the character and the location of intraneural disturbances altering the function in question is the fact that involvement of a single functional unit may occur at many different places and levels within the central nervous system. The point of implication makes a vast amount of difference in the particular functional abnormality which will be shown by the diseased unit and in the way in which the disturbances will be manifested. This phenomenon contributes much to the mystery which surrounds the activity of the central nervous system and to the confusion which is apt to arise when disease occurs, disturbing the normal neurological function.

The particular type of disorder will vary according to whether the disturbance is due to a lesion involving the effector motor cells in the nucleus, the receptive or sensory cells, the supranuclear connections with consciousness, either motor or sensory, or whether the auxiliary connections of the nucleus with other centers supplying supplementary activities are involved. Each of these possibilities can bring about a different syndrome, which must be discovered and evaluated. Any attempt to deal with this entire subject exhaustively within the limits of this paper is quite impracticable, since volumes by recognized masters in the various specialties represented in the title have been devoted to a consideration of each facet of this problem.

All of the structures mentioned in my title have been developed during the process of evolution for the purpose of identification, acquisition and ingestion of material necessary for the continued maintenance of life and, therefore, for the perpetuation of the species. In addition these anatomical formations contribute to the functions of defense or flight from inimical factors or of aggressiveness and attack when that course of action seems better suited to the circumstances. All of these structures have developed in what is termed the head-end of the organism. The eye, ear and nose have been endowed with what are called distance-receptors, representing vision, hearing and olfaction. These distance-receptors have served primarily to displace to varying distances the environment from the individual and to broaden his horizon — almost without limit in the case of vision, to very considerable distances in hearing and to some

extent where olfaction is concerned. This displacement centripitally of the environment from the individual has greatly increased his security, since he is amply forewarned as to the presence or approach of factors which threaten his existence. These functions also contribute directly to his pursuit and acquisition of food.

In the earliest phyla of the vertebrate scale, the lowly organizations representing the beginning of skeletal organization have acquired a polarity and a differentiation of a forward-moving, as contrasting with a following-after, diversification of the body and its component parts. Since the purposes of the acquisition of food and the development of means of attack and defense are most advantageously combined in the forward-moving part of the organism, these functions have been localized and developed in the head-end of the individual. Developing in this region they have established claims upon and demanded adequate cooperation from the underlying and most nearly contiguous portions of the central nervous system with the inevitable result that the forward end of the central organization develops an unparalleled condition of complexity in order to satisfy the ever-expanding demands of the outlying structures in their interrelations with all of the combinations constituting animal behavior. Other factors inherent in the nervous system itself are (1) its response to the two types of segmentation, the somatic representing the intrinsic association of skeletal or myotomic muscles with certain specific cell-groups within the central neuraxis and the branchiomeric with the gill-arch musculature and its own essential nerve cell clusters; (2) various decussations within the nervous system of the long pathways for motor control and sensory transmission and other intrinsic rearrangements and expansions, collaborate with the neural connections of these cephalic organs in making the brain-stem the most complicated and highly organized subdivision of the segmented neuraxis. The various neural structures representing the sensory and motor supplies for the eye, ear, nose and throat are all connected primarily with the brain-stem; the eye in its sensory sphere being an associated structure of the diencephalon, the interbrain, and in its motor relations connected with three of the constituent parts of the brain-stem, the mesencephalon or midbrain, the isthmus encephali and the metencephalon or pons Varolii; the nose and throat in their sensory and motor portions controlled by the metencephalon and myelencephalon, the medulla oblongata; while the ear is connected primarily with the pons Varolii. The special sense

functions of olfaction, vision and hearing are principally important from their cortical or hemispheral relations and connections.

The cerebral nerves, and therefore the structures supplied by these nerves, and the disturbances in function of these structures, serve as admirable indices of general and local involvement within the central nervous system. Many diseases of a congenital nature manifest themselves in disorders of the receptive or perceptive functions; certain diseases have a selective affinity for the conduction systems, but the chief value of the cerebral nerves lies in the anatomical situation of the nuclei which serve as receptor or effector centers within the neuraxis. The involvement of cerebral nerve nuclei acts as a guiding principle in level-diagnosis in disorders of the brain-stem, just as the various muscles and associated spinal motor-cell clusters in the spinal cord serve as indicators of level involvement. Visual defects may indicate interbrain pathological change, oculomotor defects may call attention to midbrain or pontine involvement according to their particular variety; trigeminal or facial nerve disorders designate the pons Varolii as the focus of localized disorders while involvement of the palate or tongue implicates the medulla oblongata.

The olfactory and visual apparatuses are primarily evaginations of the entire brain-wall and hence are in reality portions of the brain, displaced peripherally and are not therefore in the strict sense of the word provided with real peripheral nerves except for the fila olfactoria extending from the olfactory bulb into the olfactory mucous membrane and the branches of the ganglion-cells within the retina itself. The other nerves connected with the eye, ear, nose and throat, namely, those constituting the oculomotor apparatus, the motor and sensory supply of the nose and throat and the two portions of the auditory nerve are true peripheral nerves. This distinction constitutes a true difference, since the actual peripheral nerve supplied with a sheath of Schwann will regenerate and reinstitute function, whereas the olfactory and optic nerves not so supplied are incapable of restitution once destroyed.

In a general approach to the consideration of disorders involving the cerebral nerves, particularly those of motor and sensory function, there are certain general principles which must be kept in mind. The control of the muscles supplied by the motor cerebral nerves is of four principal kinds: (1) voluntary control; (2) associated center control; (3) reflex control; and (4) trophic control. Voluntary control is exercised by the cerebral cortex,

transmitted by the pyramidal system and delivered to the effector or motor cells of the nucleus from which the particular motor cerebral nerve emerges. The associated center controls are provided by centers of activity in the brain which contribute: automaticity with its grace and ease of movement from the basal ganglia; synergy with its accuracy and careful grading of muscular contraction from the cerebellum and other less well-known and easily-defined contributions to organized motion from other parts of the brain. Reflex and trophic control are governed by the cells of the effector nucleus itself and carry on their activities in a normal fashion when all the regulating centers are in full action or in an altered manner but still functioning if the supranuclear structures are affected by disease. If reflex and trophic control themselves disappear, then it is certain that the motor nuclei or the nerves emerging from the nuclei are involved; the normal reflexes disappear and the proper trophic influences cease. With involvement of the cortical control as well as disturbances of the nuclei or nerves, paralysis develops in both situations, a supranuclear paralysis in the first instance, an infranuclear paralysis in the second case. The characteristic, distinguishing feature between the two types of paralysis is the retention of reflex and trophic control in the former and the loss of these controls in the latter case. This differentiation is of absolute importance in determining the exact location of a lesion interfering with voluntary control. In paralysis of the limbs by reason of an involvement of the supranuclear pathways of the pyramidal tract, an abnormal stiffness and an increase in the reflexes accompany the paralysis. This does not occur for some unexplained reason in paralysis involving the pyramidal control of the cerebral nerves, the paralysis is almost without exception flaccid in character. The disturbances which appear in the activities of the eyes, the jaws, the face, lips, throat and tongue are due to the paralysis and the displacements and distortions are the results of the unopposed action of bilaterally situated and active muscles. If reflex and trophic control are retained, then one must search among the various levels of direct or associated control for the manifestations which will indicate the locus of the lesion and the structures involved by it.

The nose may be looked upon principally as a distance-receptor endowed with the functions of olfaction for the phylogenetic purpose of rendering its owner aware of factors in his environment which are attractive or repulsive, pleasing or inimical to his

existence. In the lower forms this function contributes to the protection of the herd, the opportunities for mating and for the acquisition of food. The sense of smell also provides warning of the presence of factors dangerous to the continued existence of the individual, such as forces of nature, fire and other suffocative factors or of individuals which will prey upon or devour the organism. In man, these functions have been largely reduced and smell subserves only a definitely subsidiary function in the direct appreciation of odors in the environment and its ancillary activity in the enhancement and enrichment of the sense of taste.

The nerve supply of the nose may be considered from three points of view, sensory, special sense and motor. The sensory supply of the nose, exclusive of its special sense of smell is supplied by the trigeminal nerve which sends branches not only to the exterior but also to the interior of the nose including the accessory spaces of the nasal cavities. In general, there are no particular disturbances in the central nervous system which have special significance in the alteration or disappearance of the general body sense of the nose and its associated spaces. Such disturbances are principally associated with disorders of the peripheral portions of the trigeminal nerve, either its branches or its divisions, and are recognizable by relatively simple means. One exception may be made and that is the occasional involvement of the descending root of the trigeminal nerve carrying principally pain and temperature sensibilities. This root extends downward throughout the brain-stem to the first or second cervical segments; its fibers entering the receptive trigeminal nucleus in a segmented fashion. These segments are represented by more or less roughly concentric strips of skin covering the face, scalp and upper part of the neck with the mouth as the center. Occasionally, there may be found gliotic, neoplastic, degenerative or other conditions within the brain-stem which attack the descending root of the trigeminal nerve, resulting in a loss of pain and temperature sensibility in these concentric strips of skin, thus indicating the presence within the brain-stem of pathological conditions affecting the sensory supply of the nose and mouth.

The principal importance of the nose, so far as neurological diagnosis is concerned, is provided by the sense of smell. This special sense is of definite localizing importance in intracranial diagnosis through the anatomical arrangement of the olfactory tract which lies between the ventral surface of the frontal lobe and the immediately subjacent meningeal envelopes and the

base of the anterior fossa of the skull. Neoplasms of one or both frontal lobes, meningitic processes and fractures of the base either acutely or remotely, as a result of reactive processes, may involve one or both olfactory nerves with a consequent reduction in or loss of the sense of smell in one or the other nostril. Elsberg has recently elaborated a careful and detailed method of investigating the sense of smell by the use of materials which have a maximal olfactory value and a minimal trigeminal (general body sensitivity) component; introduced into the nose in known quantities and under determined pressures, whereby a reduction in the acuity of smell may be quantitatively determined. In connection with this study, the same investigator has determined that the identification of olfactory fatigue, which may be established by a somewhat different technic, has a definitely differential significance in distinguishing between extra- and intra-cerebral lesions involving the olfactory system, in the former when the olfactory tract is involved, olfactory perception being reduced, whereas in the case of the intra-cerebral disease perception remains relatively unchanged but olfactory fatigue is materially lengthened on the side of the lesion.

Olfactory hallucinosis has long been recognized as a special symptom indicative of involvement of a special portion of the rhinencephalon, the recurved tip of the temporal lobe, the so-called uncus. Involvement of this portion of the brain, due to sclerosis, neoplasm, inflammation or other pathological factors, causes a spontaneous hallucination of odors — which are mainly of an unpleasant character, although they may on occasions present very grateful and pleasing characteristics. This olfactory symptom is usually, however, accompanied by evidence of neighborhood involvement of the brain-stem with the production of fits or localized convulsive manifestations, unilateral exophthalmos, pyramidal tract disturbances or oculomotor defects. The existence of a tumor in this region usually at some time makes its presence felt by symptoms and signs of increased intracranial pressure.

The motor manifestations of disorders of the central nervous system affecting the nose and its musculature are minimal. The nerve supplying the muscles of the nose is the facial or seventh cerebral nerve, but intra-cerebral interference with this neural component supplying the muscles of the nose is of no significant importance.

THE EYE. The eye serves as the ideal example of the distance-receptor which enables the individual to separate himself furthest from his horizon, except through the unlimited activities of thought itself. The function of the eye is the reception of stimuli of direct or reflected light conveying impressions of visual intensity and color. The function of the two eyes in mutual activity is the appreciation of form and outline and of relativity in comparison with other objects, therefore, of perspective and size. The eye also registers impressions of speed, of varying size and perspective of objects changing their positions within the visual field and, therefore, of distance through the action of convergence or divergence or conjugate movement.

The nerve supply of the eye consists of neural connections related to general body sense, special visual sense and motility. The general body sense supply of the eye is provided by the trigeminal nerve through its peripheral connections and has no particular significance in correlation with central nervous system disorders.

The special sense of vision has an extensive and important relation with the integrity of the central nervous system. The visual apparatus may be considered as undergoing its inception in the rods and cones of the retina, the visual end organs; continuing as the peripheral processes of the retinal ganglion cells, the ganglion cells themselves and the central processes which form successively the optic nerve, chiasm and tract, terminating in the primary visual centers, the pulvinar of the thalamus and the lateral geniculate body of the metathalamus. Thence the stimuli pass to the secondary receptive visual center, the striate or calcarine cortex with its associated areas of adjacent cortex for the elaboration and synthesis of visual concepts as images, playing their parts in actual vision, imagination and thought in general.

The motor innervation of the eyeball consists of the so-called oculomotor apparatus, which comprises the supranuclear, internuclear, nuclear and subnuclear or neural connections of the third or oculomotor nerve, the fourth or trochlear nerve and the sixth or abducens nerve together with that part of the oculomotor nerve which forms the midbrain autonomic outflow and supplies the two intrinsic muscles of the eyeball, the sphincter iridis and ciliary muscles. The synergistic muscle of the iris, the dilator iridis, is supplied by a branch from the ciliary ganglion of the sympathetic ganglionic system. The oculomotor nucleus is situated in the cephalic portion of the midbrain close to the

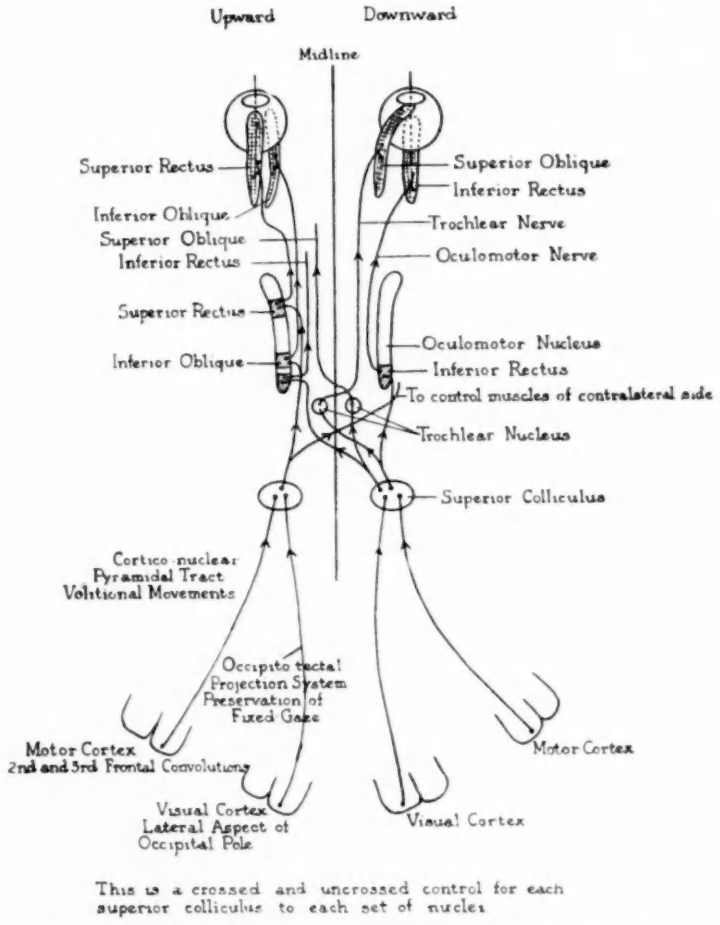


FIG. 1 — (Riley) — Mechanism for vertical deviation of gaze.

aqueduct of Sylvius and the caudal wall of the third ventricle. The trochlear nucleus is located in a similar manner somewhat caudal to the termination of the oculomotor nucleus in the isthmus encephali, the junction of the midbrain and the pons Varolii. The abducens nucleus is situated in the tegmentum of the rhombencephalon somewhat cephalad to the ponto-medullary junction. The muscles which these various nuclei control are so well known that they need not here be mentioned. The internuclear interrelations between these three nuclei are very complex and are provided by fibers of the posterior longitudinal fasciculus which

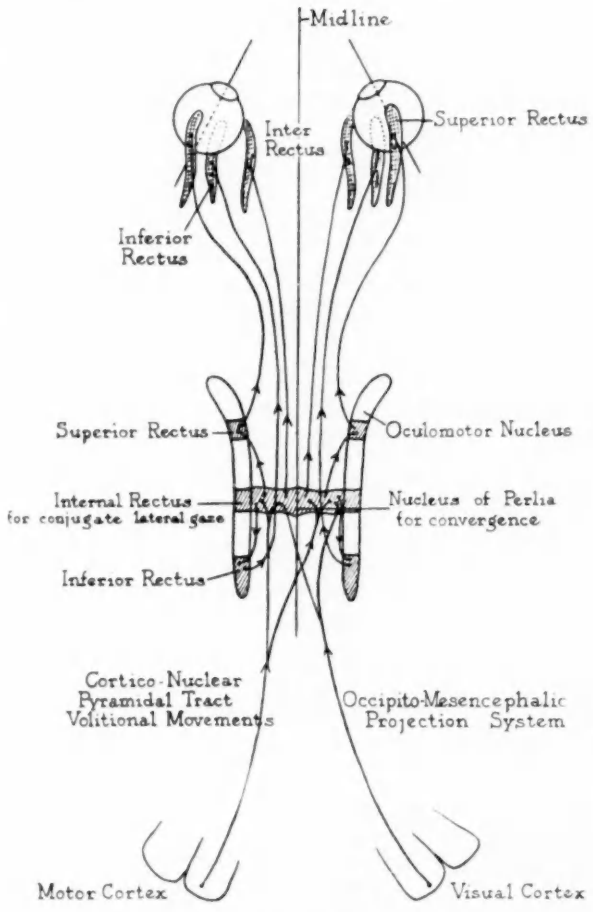


FIG. 2 — (Riley) — Mechanism for convergence.

connects the three nuclei and in which the nuclei are imbedded. The fasciculus also contains fibers which pass to the various oculomotor nuclei from the vestibular complex, and some even more primitive fiber-tracts. The supranuclear connections of the oculomotor nuclei are established by pyramidal and extrapyramidal neurones. The extrapyramidal connections are chiefly with the tectum of the midbrain for protective reflex activity, the corpus striatum which provides automatic associative control for the purpose of maintenance of tone and fluidity of movement and possibly some cerebellar connections, although these are probably

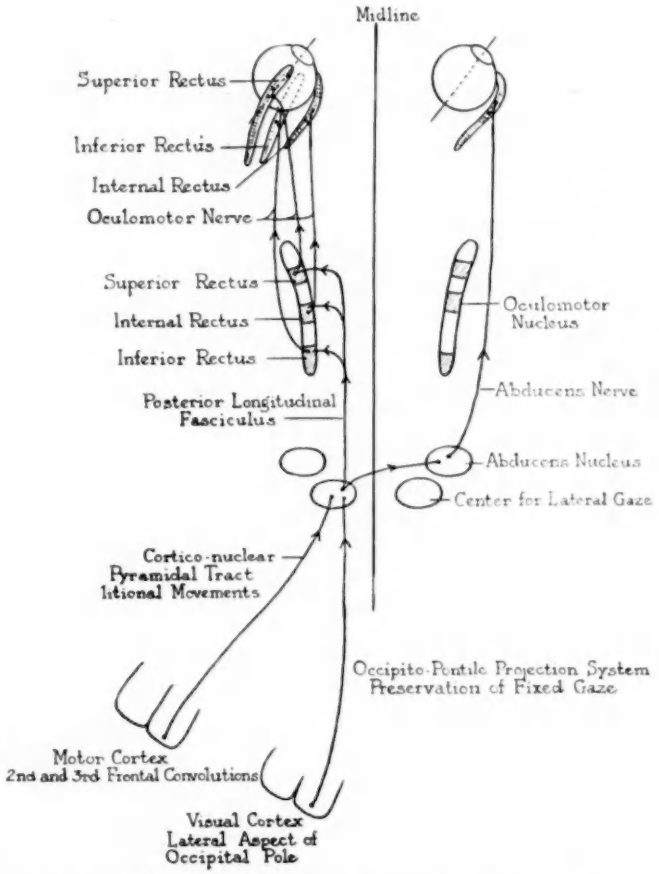


FIG. 3 — (Riley) — Mechanism for lateral deviation of gaze.

relatively insignificant since few if any purely cerebellar disorders interfere with ocular movement, and the calcarine cortex for the maintenance of fixed gaze. The pyramidal control is exerted principally from the foot of the middle frontal convolution, is contralateral in effect, the right cortex controlling left gaze and vice versa. This control is conveyed by aberrant pyramidal contingents which leave the pyramidal system in two outflows, the subthalamic, to serve principally the purposes of vertical gaze; and the peduncular contingent which separates itself from the pyramidal system in the midbrain and passes to the vicinity of the abducens nucleus to bring about the movements of lateral gaze. Other areas of the cortex are capable, upon stimulation, of

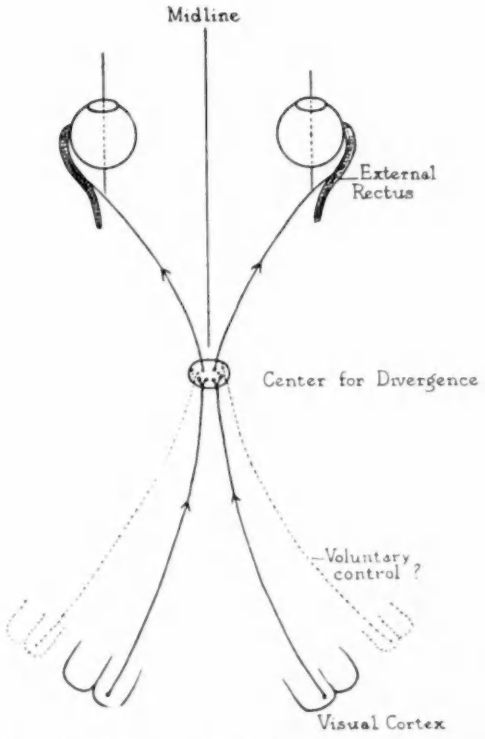


FIG. 4 — (Riley) — Mechanism for divergence.

producing ocular movements, but the principal areas of cortical control are the two right and left intermediate precentral regions in the caudal part of the middle frontal convolutions for voluntary gaze movements and the calcarine or occipital control for the preservation of fixed gaze. Interposed between the supranuclear centers and the three nuclei of the oculomotor complex are centers for the correlation and proper association of the impulses to the appropriate nuclei and nuclear segments in order to bring about the correct nuclear association for the purpose of producing conjugate movements of the two eyes along correct axes. These centers are for vertical gaze, upward and downward, for lateral gaze either right or left, for convergence and probably for divergence.

While these centers have not been satisfactorily demonstrated to exist anatomically, there is sufficient basis in clinical observation to render their existence at least probable. Patients present-

ing evidences of intact nuclear control, yet unable to produce the necessary conjugate movements, have been observed in sufficient numbers to establish a firm foundation for the supposition that such centers do exist. In addition, the theoretical necessity for such centers, wherein the proper combinations may be established whereby conjugate movements can be elaborated, is so cogent that it is highly probable, although not indubitably established, that such centers do exist.

The centers for vertical gaze may be considered to be situated in the midbrain in the vicinity of the oculomotor nucleus, probably in the superior colliculus. This center controls vertical gaze for both upward and downward movements and establishes the proper connections between homologous parts of the oculomotor nucleus of each side and between the three oculomotor nuclei so that all of them contribute to upward and downward gaze and also bring about the appropriate movement of the upper eyelids. The center for convergence is situated in the middle portion of the oculomotor nuclei adjacent to the segment controlling the internal recti. This center for convergence is called the nucleus of Perlia. The center for lateral gaze is situated in the immediate vicinity of, but not identical with, the abducens nucleus and establishes connections between the abducens nucleus of one side controlling the external rectus of one eye and the segment of the contralateral oculomotor nucleus controlling the internal rectus muscle of the other eye, so that lateral conjugate movements of the two eyes together is possible. The center for divergence is suspected on clinical grounds, but its anatomical location is as yet unknown. The effector pathways for these nuclei and centers are provided by the efferent neurones of the six nuclei. On account of the complexity of oculomotor interaction and the primary and secondary deviations produced by each ocular muscle acting alone, it is necessary in almost all conjugate movements of the eyeballs to have the entire oculomotor apparatus in continual and total interactivity.

The disturbances in vision as a special sense indicative of lesions within the central nervous system are of enormous localizing significance. In the first place, the disturbances of vision by their character almost unerringly indicate the location of the lesion if the visual disturbance is definite and clear-cut. Blindness in one eye alone, involving the entire visual field of one eye more of less completely, means an involvement of one eye or its optic nerve in front of the chiasm. In connection with blindness in

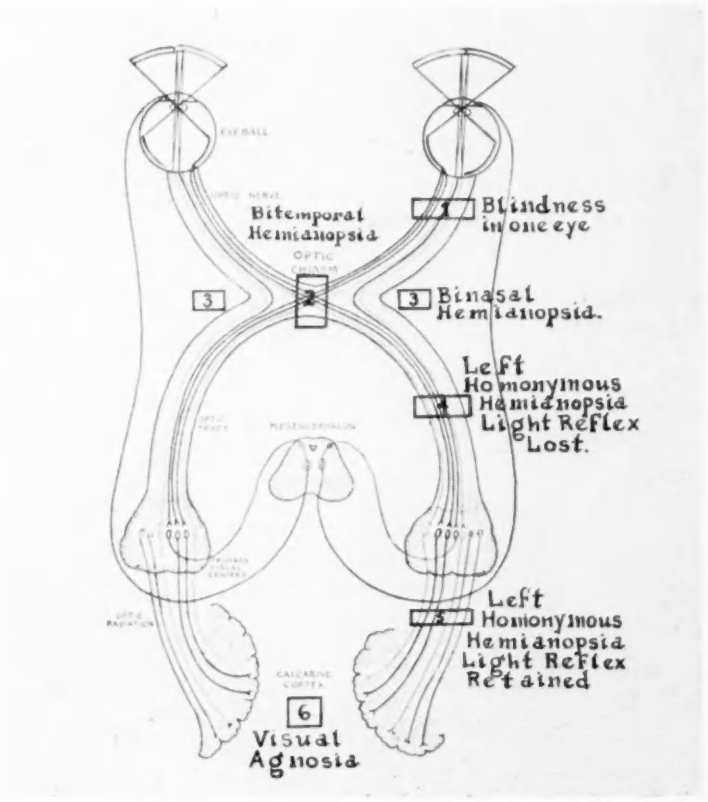


FIG. 5 — (Riley) — Diagram of the visual pathway showing the location of lesions which result in disturbances of vision.

one eye, there should always be considered the possible involvement of that particular optic nerve by a neoplasm or other lesion in the orbital gyri close to the chiasm so located as to bring pressure to bear upon the optic nerve between the optic foramen and the antero-lateral angle of the chiasm. The possibility of a meningioma arising from the sphenoid ridge (the lesser wing of the sphenoid) or from the cephalic portion of the pituitary fossa, particularly the anterior clinoid process, so affecting the nerve as to produce partial or complete loss of unilateral vision, must be kept in mind. When optic atrophy of one eye, with or without papilledema in the other eye, is associated with an upward displacement of the subcallosal and callosal fissures, as demonstrated by careful encephalography, the diagnosis of a neoplasm in this

vicinity can be made with relative certainty and attacked by the appropriate means. Involvement of the chiasm may present the various types of hemianopsia, bitemporal with the lesion located in the middle of the chiasm, binasal when the lesion affects the lateral angles of the chiasm. If the noxious moment involves the upper half of the chiasm, loss of ventral vision is the result, whereas the opposite result eventuates, if the lower half of the chiasm is involved. With post-chiasmal lesions dependent upon lesions of the central nervous system, there are presented the various types of homonymous hemianopsia, either right or left, complete or only quadrantic depending upon the extent of involvement of the optic tract. Post-chiasmal lesions situated at any point between the chiasm and the striate cortex produce the same type of visual defect with only minor differences. Lesions of the optic tract between the chiasm and the primary visual centers, the thalamic pulvinar and the lateral geniculate body are supposed to produce disappearance of both vision and the pupillary reflex to light, if the source of the illumination is confined to the blind half of the retina. Lesions affecting the optic radiation between the primary and secondary visual centers are theoretically supposed to produce a homonymous hemianopsia but with the preservation of the pupillary reflex to light if the source of illumination is confined to the blind retina. This difference is due to the fact that at the primary receptive centers, collaterals to the superior colliculi pass off to complete the pupillary reflex arc and are, therefore, unaffected by lesions situated caudal to the point of the origin of these reflex-arc fibers. This differentiation is a difficult one to make. In connection with the neural relations of the optic radiation itself, the chief point of argument at the present is the exact location of lesions responsible for the phenomenon of the split macula. Penfield and his school maintain that the retention of intact macular vision in hemianopsia is due to a sparing of a commissural band of fibers supplying macular vision, which leaves the optic radiation, crosses through the splenium of the corpus callosum and terminates in the opposite striate cortex, whereas others, including Fox and German oppose this idea of a bilateral representation of the macula and maintain that the sparing of the macula is not dependent upon the escape of this commissural bundle but upon the widespread representation of the macula in the striate cortex and the failure of the lesion to involve completely this area of cortex. The hemianopic and quadrantic losses of vision are due to local

lesions so placed anatomically as to be capable of injuring in almost an exact and mathematical fashion, the various subdivisions of the visual pathway. Neoplasms, cysts, vascular lesions, aneurysms, inflammations, cerebral traumata and large patches of sclerosis are the principal noxious elements which produce these visual defects. It must be remembered that extra-hemispheral neoplasms and other factors may produce involvement of the optic tract, such as pontine neoplasms which compress the optic tract between itself and the mesial aspect of the hemisphere as the tract sinks into the cerebral substance. In such instances a homonymous hemianopsia may be associated with symptoms which can arise only from pontine lesions such as nuclear facial or trigeminal involvement, the syndrome presenting a puzzling picture unless the exact anatomical relations are constantly borne in mind.

The further implications of cortical involvement of the associative visual areas need only be mentioned as mind-blindness, visual agnosia, as indicative of cortical disturbances. Visual hallucinosis is frequently seen in general cerebral disease, the psychoses, and with irritative lesions affecting the visual tract and the striate and associative areas of the cortex. In brief, the formed hallucinations seem to be associated more with the tract implications, whereas the unformed hallucinations, contrary to one's expectation, are relatively more frequent with striate cortex involvement.

The retinal changes associated with intrinsic disease of the central nervous system, such as retinitis pigmentosa, primary hereditary axial neuritis, and amaurotic family idiocy, should be borne in mind.

The implications of papilledema, primary or secondary optic atrophy, temporal pallor and other disturbances of a similar kind with intrinsic general or local brain disease merit special attention.

The factors producing disturbances in the oculomotor apparatus may exist at any level of the nervous system from the cortex to the neuromuscular junction. The various types of interference with oculomotor activity depend for their effects upon the structures implicated and upon the exact anatomical location of the pathological lesion. Lesions of the frontal cortex and the aberrant pyramidal contingents produce gaze disturbances which are always conjugate and therefore affect both eyes. These disturbances affect principally lateral gaze either to the right or to the left, the patient being unable to turn the eyes to the right or to

the left according to the system involved. This type of disturbance is seen mainly in cortical or subcortical lesions principally on a vascular basis and often associated with a hemiplegia. The cortical control is contralateral, the left cortex governing movements of the two eyes to the right. Under these circumstances passive rotation of the head away from the direction of paralyzed gaze with the eyes fixed upon a distant object may sometimes result in the movement of the eyes into the zone of paralyzed voluntary movement, indicating the continued activity of the mechanism for fixed gaze which is situated in the occipital cortex and is independent of purely volitional control and of the lesions producing voluntary paralysis.

Paralysis of vertical gaze is more frequently seen in involvement of the subthalamic contingent of the aberrant pyramidal system for vertical gaze in lesions of the thalamus and subthalamus by neoplastic, traumatic or inflammatory disease. This type of paralysis is often observed in lesions, principally pineal neoplasms, which affect the superior colliculi and thus, probably, the center for vertical gaze. Forced movements, either tonic or clonic, of upward, downward or lateral gaze, are seen in jacksonian seizures and in the ocular crises of epidemic encephalitis. Paralysis of convergence is often seen in epidemic encephalitis due probably to an involvement of the center for convergence. Paralysis of divergence, due in all probability to an involvement of the hypothetical center for divergence, has been frequently recorded but not ascribed to any particular etiological process. Lesions of the posterior longitudinal fasciculus produce dissociation of conjugate gaze through an interruption of the internuclear connections which make possible conjugate movements of the eyeballs. These lesions result in uncoordinated, wild, bizarre movements of the eyes when conjugate movements are attempted.

Disturbances in oculomotor control due to interference with the extrapyramidal systems are relatively few in number. The fixity of gaze in paralysis agitans and that found in chronic encephalitis is well known. Intrinsic cerebellar lesions, as a rule, do not produce oculomotor asynergia; cerebellar lesions involving the vestibular part of the cerebellum and vestibular lesions themselves either of the peripheral, conductive, receptive, or associative portions of the vestibular nerve, as well as several other conditions, are prone to produce nystagmus. Occasionally, abnormal involuntary movements of the eyeballs are seen in infectious chorea, athetosis, multiple sclerosis and other diseases.

Rhythmic oscillatory movements of the eyes usually associated with similar movements in the palate, pharynx and other regions have been frequently reported in a peculiar degenerative condition involving the central tegmental tract, inferior olivary nucleus, and the dentate nucleus of the cerebellum.

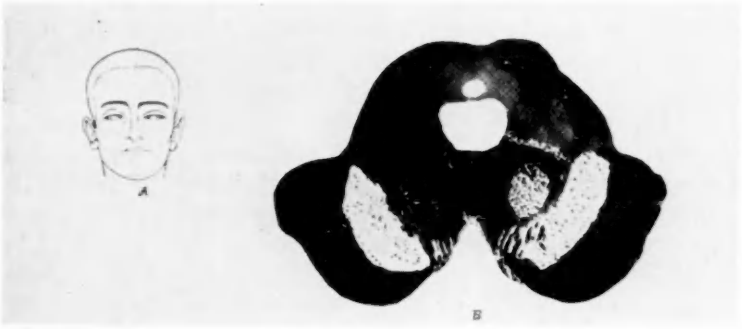
The other disturbances of the oculomotor apparatus depend upon infranuclear lesions and result from the involvement of the oculomotor, trochlear or abducens nerves, wholly or in part. The resultant paralysis affects a single muscle in the case of the fourth and sixth nerves and either an isolated muscle or combined muscles depending upon the extent of the lesion in the case of the oculomotor nerve. The paralysis is of the muscle involved, the eye assuming a deviated position on account of the unopposed activity of the synergistic unparalyzed muscles. If the sixth nerve is involved, the movement of the eye outward is lost, an internal strabismus resulting. The deviation of the eye is not so definite with paralysis of the superior oblique muscle, but the eye tends to be displaced upward and outward. In the case of involvement of the oculomotor nerve, the upper eyelid falls, producing a ptosis, and the eyeball is deviated outward due to the unopposed traction of the external rectus muscle. Paralysis of a nuclear or infranuclear character produces a loss of parallel fixation of the optical axes and a diplopia results.

The exact localization of lesions in the central nervous system must be made both longitudinally and transversely. The various cerebral nerves serve to indicate the longitudinal position of lesions within the brain-stem. In involvement of the brain-stem the grouping of other symptoms resulting from the implication of other anatomical structures indicates the transverse location and extent of the particular lesion. Knowledge of the finer anatomy of the brain-stem is necessary in order to solve these neurological cross-word puzzles. The elements of neurological diagnosis, under these circumstances, are too numerous and elaborate to be retained by only casual contact with the anatomy and the clinical evidences of disturbed function and only the neurologist can be expected to be sufficiently familiar with the elements of brain-stem diagnosis to be able to make a diagnosis in these intricate situations. The anatomical and physiological constituents of the brain-stem are associated together in certain well-known patterns which vary in each successive level of the neuraxis. Tracts alter their position in various levels of the central

nervous system and, therefore, the pattern of neurological disorders will be altered in successive levels.

The oculomotor apparatus is the level-indicator of lesions occurring in the midbrain and pons Varolii. In the midbrain, lesions are responsible for involvement of the oculomotor nucleus or the centers for vertical gaze or convergence. Disturbances of the trochlear nucleus indicate the implication of the isthmus encephali, while disorders of the abducens nucleus or the center for lateral gaze cast suspicion on the metencephalon as the site of the lesions.

On account of the fact that vascular accidents present a very definite tendency to occur at certain particular places in the nervous system, the repeated development of similar symptom-pictures has resulted in the recognition of certain syndromes which will usually present an identical or closely related neurological pattern. These syndromes are fairly stereotyped and have received names which have made them familiar phenomena to the neurologist. Syndromes have been recognized in all levels of the brain-stem, being particularly numerous in the midbrain and medulla oblongata. While the exact cause for the frequent and almost identical recurrence of the syndromes is not clear, it is certainly associated specifically with the terminal or end-artery character of the blood supply of the neuraxis and generally with



From TILNEY and RILEY: *"The Form and Functions of the Nervous System"*

FIG. 6 — (Riley) — *A.* Syndrome of the oculomotor nuclei. Syndrome of the mesencephalic central gray matter. Bilateral external and internal ophthalmoplegia (paralysis of the eye muscles) of the oculomotor type.

B. Cross section through the midbrain showing the location of the lesion in the *syndrome of the mesencephalic central gray matter:* Involvement of both oculomotor nuclei.

the length, size, angulation and lack of firm support by the neural tissues of the arteries of the brain-stem.

The syndromes which depend for their localization upon an involvement of the oculomotor apparatus and associated structures are as follows:

1. *The Syndrome of the Mesencephalic Central Gray Matter.* (See Fig. 6). This syndrome is due to involvement of the blood vessel or vessels which supply the gray matter of the aqueduct of Sylvius and, therefore, the third nerve nuclei. This supply is frequently provided by a single unpaired vessel which penetrates the tegmentum from the arteries at the ventral surface.

The constituent symptoms of this syndrome are:

a. Bilateral internal and external oculomotor ophthalmoplegia due to involvement of both oculomotor nerves.

b. Divergent strabismus due to the unopposed action of both external recti through the abducens nuclei.

c. Bilateral ptosis due to paralysis of the levator palpebri superioris muscles.

2. *The Syndrome of Superior Alternating Hemiplegia: the Syndrome of Weber* (See Fig. 7). This syndrome results from the involvement of a vessel which supplies the median portion of one-half of the basis of the neuraxis when the blood supply is provided by a paired group of blood vessels. This vessel supplies the ventral structures, the pyramid and the oculomotor nerve. This syndrome may also be produced by hemorrhage, neoplasm or injury of this part of the neuraxis.

The constituent symptoms of this syndrome are:

a. Homolateral

Internal and external oculomotor ophthalmoplegia due to involvement of the homolateral oculomotor nerve with ptosis

b. Contralateral

Upper motor neurone hemiplegia due to involvement of the pyramidal tract evidenced by paralysis, spasticity and increased reflexes.

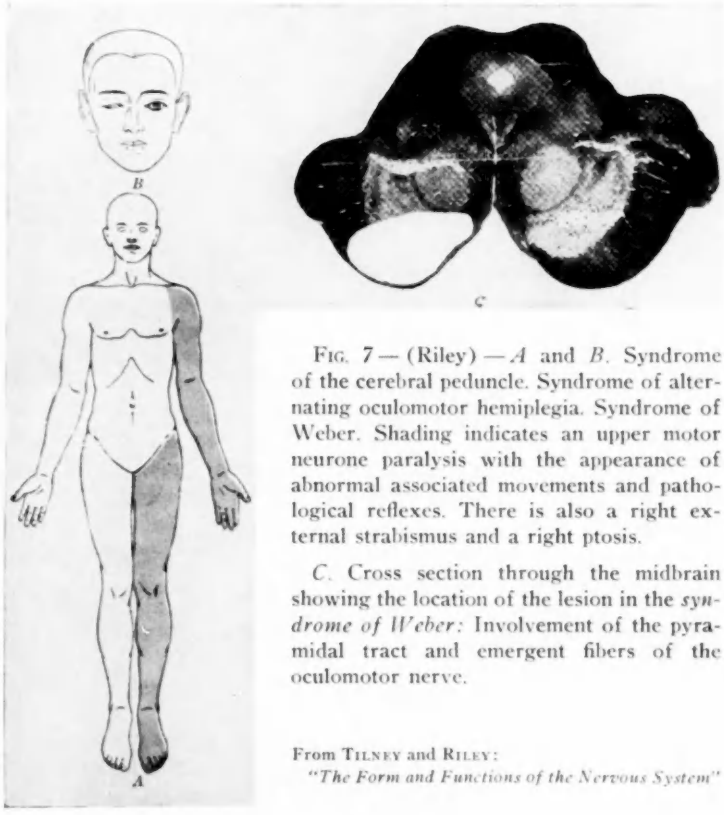


FIG. 7 — (Riley) — *A* and *B*. Syndrome of the cerebral peduncle. Syndrome of alternating oculomotor hemiplegia. Syndrome of Weber. Shading indicates an upper motor neurone paralysis with the appearance of abnormal associated movements and pathological reflexes. There is also a right external strabismus and a right ptosis.

C. Cross section through the midbrain showing the location of the lesion in the *syndrome of Weber:* Involvement of the pyramidal tract and emergent fibers of the oculomotor nerve.

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3. *The Syndrome of the Mesencephalic Tegmentum: the Syndrome of Benedict* (See Fig. 8). This symptom-picture is usually produced by an involvement of one-half of the tegmentum by occlusion of the vessels entering the neuraxis for the lateral surface. The oculomotor nerve, the red nucleus and the mesial fillet are involved.

The constituent symptoms of this syndrome are:

a. Homolateral
   Internal and external oculomotor ophthalmoplegia due to involvement of the homolateral oculomotor nerve with ptosis.

b. Contralateral
   (1) Increased tone and tremor due to involvement of the red nucleus;

(2) Hemianæsthesia due to involvement of the sensory tracts conveying sensations of pain and temperature, touch, and the deep sensibilities of position and movement, spatial discrimination, localization of the area of cutaneous surface stimulated and loss of vibratory sensibility.

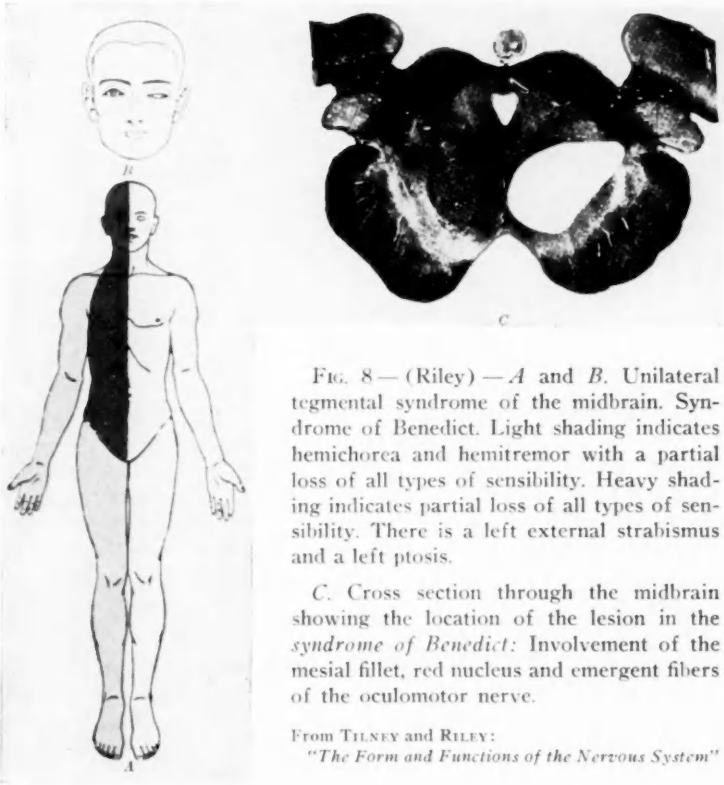

FIG. 8 — (Riley) — *A* and *B*. Unilateral tegmental syndrome of the midbrain. Syndrome of Benedict. Light shading indicates hemichorea and hemitremor with a partial loss of all types of sensibility. Heavy shading indicates partial loss of all types of sensibility. There is a left external strabismus and a left ptosis.

C. Cross section through the midbrain showing the location of the lesion in the *syndrome of Benedict:* Involvement of the mesial fillet, red nucleus and emergent fibers of the oculomotor nerve.

From TILNEY and RILEY:
"The Form and Functions of the Nervous System"

4. *The Syndrome of the Interpeduncular Space* (See Fig. 9). This syndrome may result from inflammatory, vascular, neoplastic or traumatic causes. The point of implication is the interpeduncular space and the structures involved are those which are found in this space, the two pyramidal tracts and oculomotor nerves.

The constituent symptoms of this syndrome are:

a. Bilateral external and internal oculomotor ophthalmoplegia with bilateral ptosis due to involvement of both oculomotor nerves.

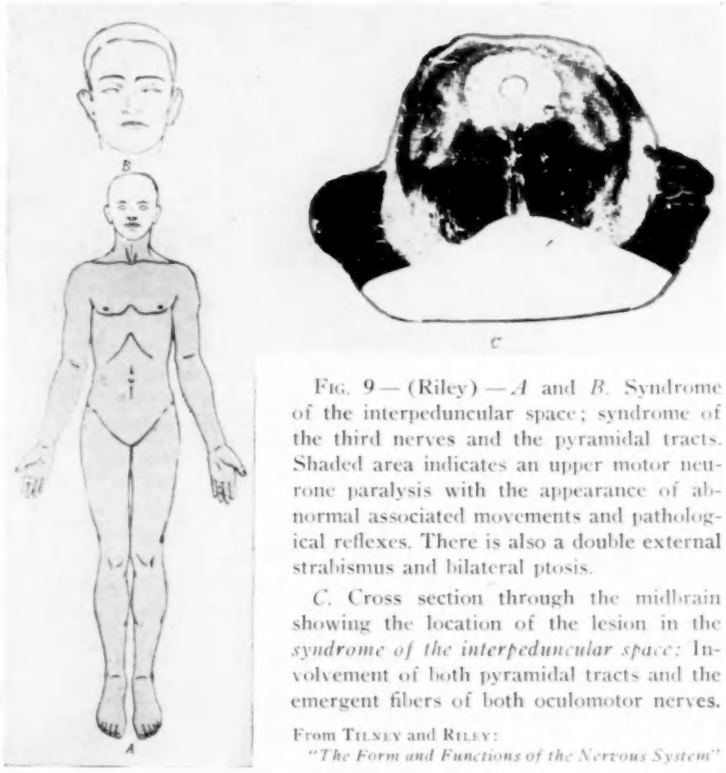

FIG. 9 — (Riley) — *A* and *B*. Syndrome of the interpeduncular space; syndrome of the third nerves and the pyramidal tracts. Shaded area indicates an upper motor neurone paralysis with the appearance of abnormal associated movements and pathological reflexes. There is also a double external strabismus and bilateral ptosis.

C. Cross section through the midbrain showing the location of the lesion in the *syndrome of the interpeduncular space:* Involvement of both pyramidal tracts and the emergent fibers of both oculomotor nerves.

From TILNEY and RILEY:
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b. Bilateral upper motor neurone tetraplegia with paralysis, spasticity and increased reflexes due to the bilateral involvement of the pyramidal tracts.

The preceding are the principal syndromes which have their anatomical focus in the mesencephalon and demonstrate their presence by interference with the function of the third cerebral nerves.

The next succeeding cerebral nerve nucleus in the brain-stem is that of the fourth cerebral nerve, the trochlear nerve which is situated in the isthmus encephali. Isolated intraxial involvement of this nucleus is rarely seen and it is problematical whether implications of this nucleus within the neuraxis can with certainty be distinguished from involvement of the trochlear nerve in its peripheral course.

The pons Varolii is richly endowed with cerebral nerve nuclei which by their implication are of aid in indicating various levels of involvement of this segment of the brain-stem.

In the dorsal part of the pons Varolii, the abducens nucleus and the center for the lateral gaze are closely associated with the facial nerve in its peculiar intraxial course. Limited lesions in this region may produce homolateral external rectus palsy or a facial paralysis either singly or in combination.

Involvement of the abducens nucleus or nerve indicates longitudinally the metencephalon or pons Varolii as the site of the lesion. The particular pattern of associated neurological disorders demonstrates the transverse location and extent of the pathological disorder.

In addition to the symptoms indicating involvement of the cerebral nerves or nuclei, the mesial fillet conveying discriminative sensibility, the spino-thalamic tracts conducting impulses of pain and temperature, the pyramidal tracts controlling voluntary motion and the cerebellar connections governing coordination and synergy may be involved by vascular, inflammatory or traumatic agencies, resulting in the symptom-pictures which constitute the syndromes of the pons Varolii.

The oculocephalogyric pathways may be involved in the pons Varolii without direct involvement of the abducens nerve or nucleus. This type of disturbance results in an inability either complete or partial on the part of the patient to turn the head and eyes in one direction or another, the eyes being turned into the opposite position of conjugate deviation due to the unopposed action of the antagonistic oculocephalogyric impulses. The exact direction of paralysis of gaze, head movement and of the forced conjugate deviation will depend upon whether the oculocephalogyric pathway is involved at the level of the center for these movements or before the pathway crosses the midline to end in the center. If the lesion affects the oculocephalogyric pathway above its point of crossing, the paralysis of gaze and head movement will be on the side of the body opposite the location of the lesion and the deviation due to the unopposed antagonist will be toward the side of the lesion. If the center be involved or the oculocephalogyric pathway involved after its point of crossing the paralysis of gaze and head movement will be on the same side as the lesion and the head and eyes will be turned away from the side of the lesion.

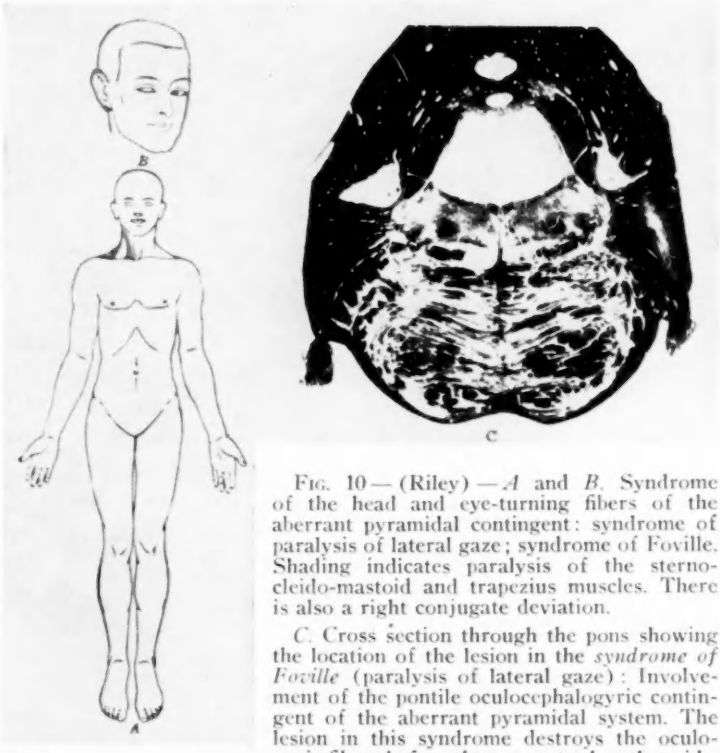

FIG. 10 — (Riley) — *A* and *B*. Syndrome of the head and eye-turning fibers of the aberrant pyramidal contingent: syndrome of paralysis of lateral gaze; syndrome of Foville. Shading indicates paralysis of the sterno-cleido-mastoid and trapezius muscles. There is also a right conjugate deviation.

C. Cross section through the pons showing the location of the lesion in the *syndrome of Foville* (paralysis of lateral gaze): Involvement of the pontile oculocephalogyric contingent of the aberrant pyramidal system. The lesion in this syndrome destroys the oculogyric fibers before they cross to the other side, and also the uncrossed cephalogyric fibers, so that a single lesion paralyzes the levogyric oculomotor apparatus and the right cephalogyric muscles.

From TILNEY and RILEY: *"The Form and Functions of the Nervous System"*

1. *The Syndrome of the Oculocephalogyric Control: the Syndrome of Foville* (Fig. 10).

The constituent symptoms of this syndrome are:

a. Paralysis of conjugate movement of the eyes.
b. Paralysis of movement of the head in agreement with the gaze paralysis.
c. Conjugate deviation of the eyes away from the direction of paralyzed gaze.

The direction of gaze and head-turning paralysis will vary in accordance with the exact location of the lesion.

In the illustration, the eyes are drawn to the right and gaze and head turning movements are paralyzed toward the left, therefore, the lesion as shown in the ventro-mesial region of the left side of the tegmentum affects the oculocephalogyric movements above the point of crossing of the oculogyric fibers.

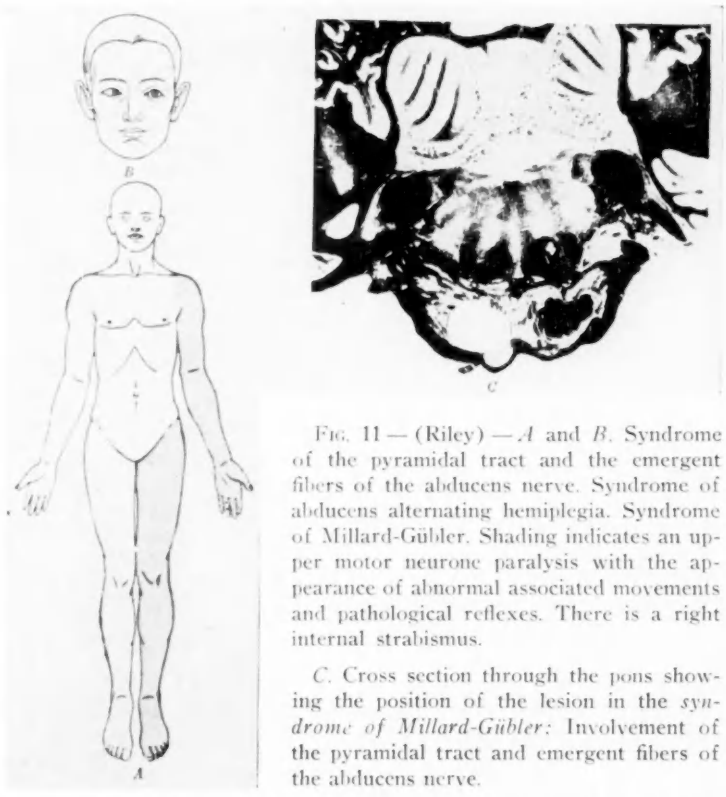

FIG. 11 — (Riley) — *A* and *B*. Syndrome of the pyramidal tract and the emergent fibers of the abducens nerve. Syndrome of abducens alternating hemiplegia. Syndrome of Millard-Gübler. Shading indicates an upper motor neurone paralysis with the appearance of abnormal associated movements and pathological reflexes. There is a right internal strabismus.

C. Cross section through the pons showing the position of the lesion in the *syndrome of Millard-Gübler:* Involvement of the pyramidal tract and emergent fibers of the abducens nerve.

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If the pathological process is situated ventrally on the surface and fairly close to the midline, the structures involved will be the abducens nerve and the pyramidal tract resulting in

2. *The Syndrome of Middle Alternating Hemiplegia: the Syndrome of Millard-Gübler* (Fig. 11).

The constituent symptoms of this syndrome are:

a. Homolateral
   Paralysis of the external rectus with an internal strabismus due to involvement of the abducens nerve.

b. Contralateral
   Hemiplegia with paralysis, spasticity and increased reflexes due to involvement of the pyramidal tract.

If the pathological process extends somewhat laterally, the facial nerve will be implicated, adding to the homolateral abducens palsy, an involvement of all of the muscles of one side of the face, either completely or partially, a so-called lower or peripheral type of facial palsy.

If the lesion extends somewhat more dorsally, the syndrome presented will be the same as the preceding, but with the addition of symptoms due to an involvement of the mesial fillet; the lesion affecting the mesial part of the tegmentum and basis. This syndrome usually results from vascular accidents principally thrombotic ones in this region and is called:

3. *The Syndrome of Middle Alternating Hemianaesthetic Hemiplegia.*

The constituent symptoms of this syndrome are:

a. Homolateral
   Paralysis of the external rectus muscle due to involvement of the abducens nerve.

b. Contralateral
   (1) Hemianæsthesia affecting the deep sensibilities with loss of the sense of position and movement, spatial discrimination, localization of the area of cutaneous surface stimulated and loss of vibratory sensibility due to involvement of the mesial fillet.
   (2) Upper motor neurone hemiplegia with paralysis, spasticity and increased reflexes due to involvement of the pyramidal tract.

At times, the pathological process may fail to involve the mesial fillet but does implicate the aberrant pyramidal bundles supplying the function of lateral gaze, together with the pyramidal tract. This combination results in the production of:

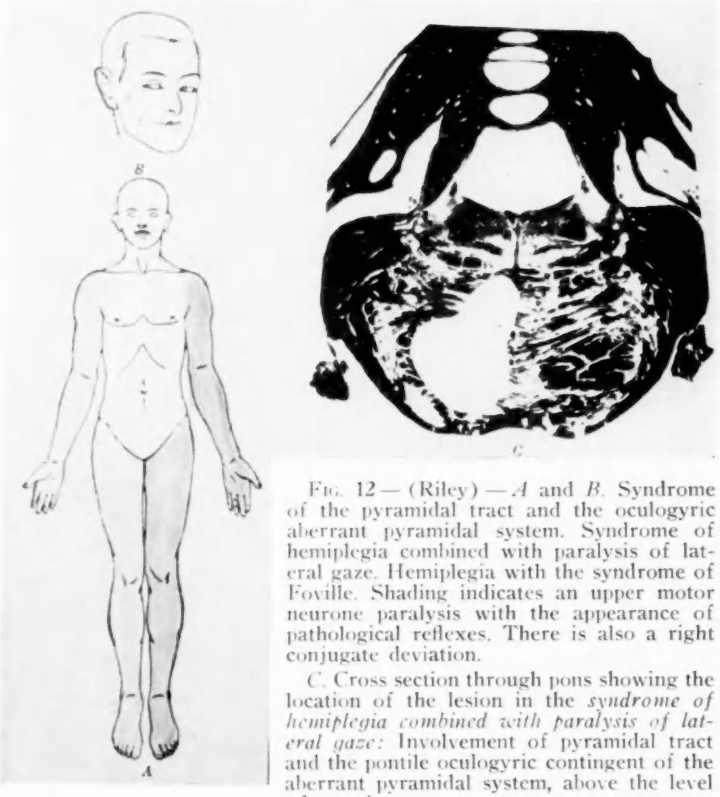

FIG. 12 — (Riley) — *A* and *B*. Syndrome of the pyramidal tract and the oculogyric aberrant pyramidal system. Syndrome of hemiplegia combined with paralysis of lateral gaze. Hemiplegia with the syndrome of Foville. Shading indicates an upper motor neurone paralysis with the appearance of pathological reflexes. There is also a right conjugate deviation.

C. Cross section through pons showing the location of the lesion in the *syndrome of hemiplegia combined with paralysis of lateral gaze:* Involvement of pyramidal tract and the pontile oculogyric contingent of the aberrant pyramidal system, above the level of crossing.

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4. *The Syndrome of Foville with Hemiplegia* (Fig. 12).

The constituent symptoms of this syndrome are:

a. An upper motor neurone hemiplegia with increased reflexes, paralysis and spasticity.

b. Oculogyric paralysis, the lesion being above the level of crossing and producing a paralysis of gaze away from the side of the lesion.

c. Conjugate deviation toward the side of the lesion from the unopposed action of the antagonistic oculomotor mechanism.

Slight extension dorsally and laterally of the lesion includes the mesial fillet in the group of structures involved with the ad-

dition of a loss of discriminative sensibility on the half of the body opposite the side of the lesion. This combination of symptoms represents:

5. *The Syndrome of Hemianaesthetic Hemiplegia with the Syndrome of Foville* (Fig. 13).

The constituent symptoms of this syndrome are:

a. A contralateral upper motor neurone hemiplegia with paralysis, spasticity and increased reflexes due to an involvement of the pyramidal tract above the decussation.

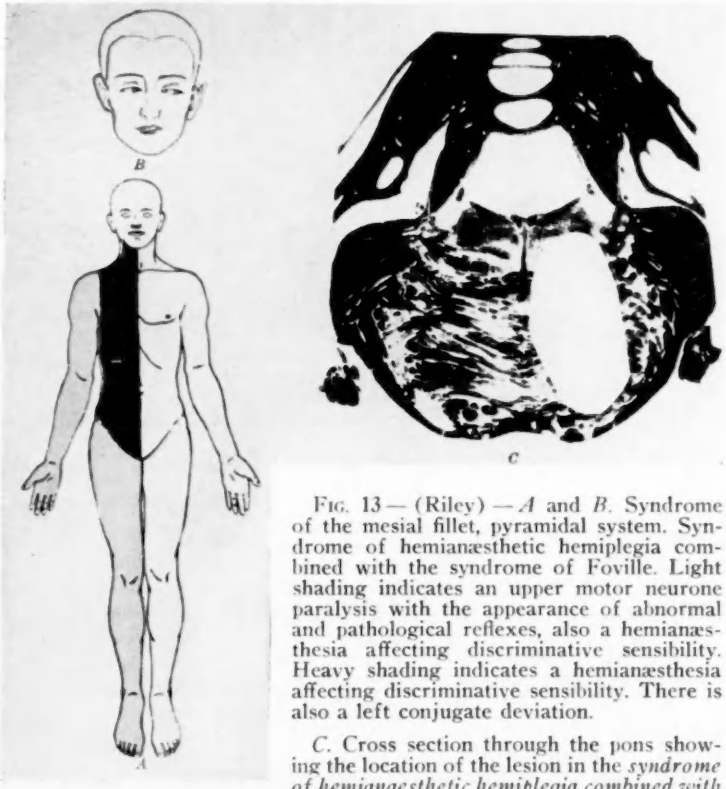

FIG. 13 — (Riley) — *A* and *B*. Syndrome of the mesial fillet, pyramidal system. Syndrome of hemianæsthetic hemiplegia combined with the syndrome of Foville. Light shading indicates an upper motor neurone paralysis with the appearance of abnormal and pathological reflexes, also a hemianæsthesia affecting discriminative sensibility. Heavy shading indicates a hemianæsthesia affecting discriminative sensibility. There is also a left conjugate deviation.

C. Cross section through the pons showing the location of the lesion in the *syndrome of hemianaesthetic hemiplegia combined with a paralysis of lateral gaze:* Involvement of pyramidal tract, mesial fillet, and oculogyric contingent of aberrant pyramidal system above the level of crossing.

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b. A contralateral hemianæsthesia affecting the deep sensibilities with loss of the sense of position and movement, spatial discrimination, localization of the area of the cutaneous surface stimulated and loss of vibratory sensibility due to involvement of the mesial fillet.

c. Oculogyric paralysis due to involvement of the oculogyric aberrant pyramidal system for lateral gaze above the level of crossing producing a paralysis of gaze away from the side of the lesion and a conjugate deviation toward the side of the lesion.

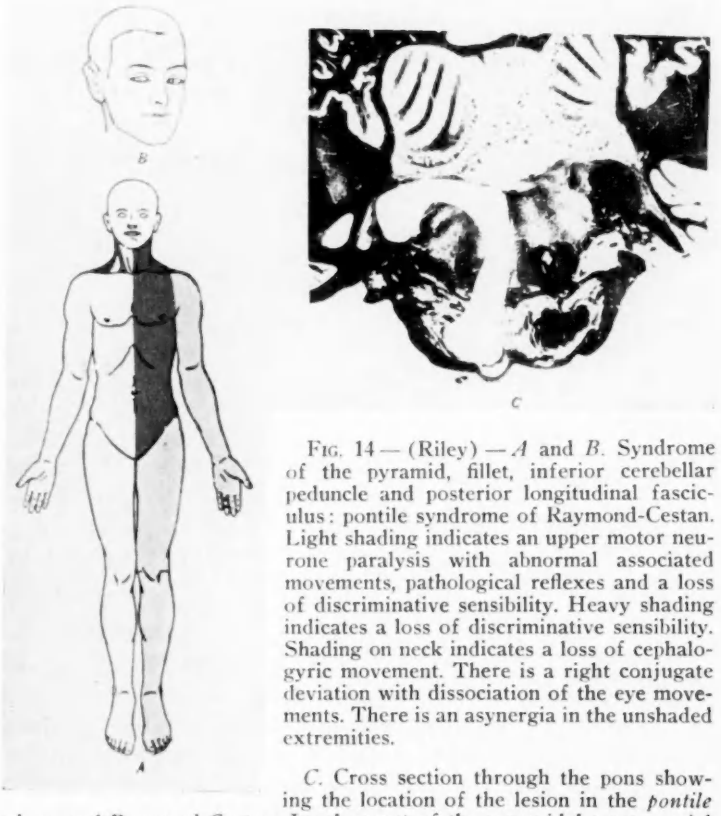

FIG. 14 — (Riley) — *A* and *B*. Syndrome of the pyramid, fillet, inferior cerebellar peduncle and posterior longitudinal fasciculus: pontile syndrome of Raymond-Cestan. Light shading indicates an upper motor neurone paralysis with abnormal associated movements, pathological reflexes and a loss of discriminative sensibility. Heavy shading indicates a loss of discriminative sensibility. Shading on neck indicates a loss of cephalogyric movement. There is a right conjugate deviation with dissociation of the eye movements. There is an asynergia in the unshaded extremities.

C. Cross section through the pons showing the location of the lesion in the *pontile syndrome of Raymond-Cestan:* Involvement of the pyramidal tract, mesial fillet, posterior longitudinal fasciculus and the inferior cerebellar peduncle.

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An extensive vascular lesion of the pons Varolii may take place and combine the characteristics of several of the preceding syndromes with the addition of cerebellar symptoms as a result of the lesion extending dorso-laterally to involve the inferior cerebellar peduncle. The involvement of the posterior longitudinal fasciculus accounts for the oculocephalogyric paralysis. This group of symptoms is:

6. *The Pontile Syndrome of Raymond-Cestan* (Fig. 14).

The constituent symptoms of this syndrome are:

a. Contralateral
   (1) Hemiplegia with the usual symptoms resulting from involvement of the pyramidal tract.
   (2) Hemianæsthesia with a loss of discriminative sensibility as a result of a disturbance in sensory conduction by the mesial fillet.
   (3) Oculocephalogyric paralysis for movements away from the side of the lesion due to a lesion of the oculocephalogyric fasciculus.

b. Homolateral
   Asynergia, incoordination and ataxia of the extremities due to involvement of the inferior cerebellar peduncle.

These are the principal syndromes of the brain-stem which involve the nuclei of the cerebral nerves connected with the oculomotor apparatus. This list does not by any means exhaust the possibilities of involvement, especially by scattered lesions throughout the brain-stem. This group is, however, sufficiently inclusive to represent the more usual syndromic types of nuclear involvement of the constituents of the oculomotor apparatus.

THE EAR. The twin functions of the ear are equilibration and audition. Of the two, the vestibular activity is much the older, phylogenetically, antedating by many thousands of years the development of the sense of hearing. A perfectly functioning equilibratory system is found in even the lowest of the vertebrate phyla, whereas hearing only appears as a special function with the assumption of terrestrial life. in the aquatic forms, the equilibratory apparatus is much more highly specialized than in the younger additions to the vertebrate line, and presents a system of structures distributed on the surface in the form of the lateral

line organs which register varying impressions of pressure exerted by the homogeneous aqueous environment. This equilibratory system composed of the membranous labyrinth and the lateral line organs is served by the vestibular nerve and its central organizations. With the assumption of terrestrial life, this system of lateral line organs recedes and the central constituents are taken over by the newly developing function of hearing which reconstructs the central apparatus and develops out of it, the cochlea. The neural connections of the cochlea elaborate the cochlear nerve and nuclei, and the central nervous system constructs a new set of complicated central connections which in the orderly processes of evolution and cephalization finally reach the pallium. In the course of the upbuilding of the acoustic function, many levels develop which provide an ever-increasing amplitude for auditory associations until the final product is achieved in the highly specialized cortical receiving stations of the transverse gyri of Haeschl and the adjacent cortex for the elaboration, systematization, and associative purposes of mental activity. The successive levels which are clearly evident in the anatomical arrangement of the auditory apparatus are the receptive nuclei of the medulla, the inferior colliculi, the mesial geniculate bodies and finally the cortical receptive areas in the superior temporal convolutions. Each one of these levels serves its purpose and becomes relegated to the background as the process of telencephalization expands and complicates the central nervous organization. Consequent upon this gradual upbuilding of the acoustic function, the bilateral representation of audition becomes more and more fully developed, and, as a result, but few if any strictly unilateral disorders of the central nervous system are recognizable through interference with the acoustic pathways.

The auditory complex develops primarily as an outgrowth of the outer wall of the primitive rhombencephalon, the auditofacial groove, but this process is not carried to the extent which is seen in the olfactory and optic apparatuses and the receptive nuclei of the auditory complex remain incorporated in the central nervous system. The receptive apparatus evolves from the otic vesicle, an invagination of the ectoderm which in the early stages establishes connection with the neural outgrowth. The otic vesicle gives rise to the membranous labyrinth and cochlea.

The auditory complex from its inception serves special functions, first of equilibration, later of audition, and it has never had any general sensory or motor components.

The vestibular function remains essentially as a subpallial elaboration. It does, however, establish perfectly definite cerebellar connections but exact evidence of pallial representation is still wanting. That there are connections with consciousness is perfectly evident in the normal sensations of vertigo and the occasional fairly definite vertiginous sensations connected with cerebral disease. The temporal and closely adjacent areas of the parietal lobe seem to be the principal loci which are capable of originating these sensations as a result of pathological processes arising in them. The cerebellar connections are provided by way of the juxta-restiform body, lying mesial to the restiform body, the two constituting the main component structures of the inferior cerebellar peduncle. The vestibulo-cerebellar fibers end in the fastigial or roof nuclei, constituents of the intrinsic nuclei of the cerebellum, and also present connections with the cerebellar vermis. These equilibratory centers were originally situated in the neuraxis, but became incorporated in the cerebellum as it developed its primary equilibratory function for the lower vertebrate phyla. The later and much more evident function of the cerebellum as the great controller of coordination and collaborator in the organization of the skilled acts is only seen as the pallium develops. The cerebellum fundamentally is the organ of equilibration. The expansion of the cerebellum as a coordinative center takes place *pari passu* with the evolution of the cerebral hemispheres. The cortex, the pontine nuclei, the middle cerebellar peduncle and the lateral hemispheres of the cerebellum develop as a closely integrated system, each one dependent upon the other. System disease of this part of the central nervous organization discloses itself as a process affecting all of these units, each one undergoing proportionate and equivalent involvement. The neural disorders of equilibration depend principally upon the involvement of the labyrinth and the conduction system, the vestibular nerve, and are, therefore, outside the scope of this consideration. The disorders of the equilibratory apparatus dependent upon central nervous lesions are to a considerable extent limited to pathological processes in close contiguity with the neuraxis, in the cerebello-pontine angle and in the brain-stem. These disorders constitute the central neural contributions to the syndrome of Ménière, resultant upon irritative processes affecting the conductive and central receptive structures of the vestibular complex, namely, the vestibular nuclei of the medulla oblongata and pons Varolii. Central lesions, such as tumors, inflammation, vascular

lesions and localized sclerotic or other degenerative processes, may give rise to intractable aural vertigo which is resistent to all of the usually recognized methods of treatment. In the region of the receptive vestibular nuclei is situated a plexus of veins which frequently by engorgement and the production of varices gives rise to persistent and abnormal stimulation and irritation of the vestibular apparatus resulting in the symptoms of vertigo, loss of equilibrium, and its associated nausea. Often on account of the close contiguity of the acoustic centers, tinnitus and partial unilateral deafness may accompany the equilibratory disturbances. As already stated, central lesions in the temporal and parietal regions are also capable of giving rise to vertigo and other disturbances of equilibration, namely, the various types of pulsion — anteropulsion, retropulsion and lateropulsion. These disturbances are also frequently accompanied by nausea. Exact knowledge in regard to the localization of these disorders is lacking.

Many otologists have attempted to split up the pathways in the neuraxis in accordance with the various portions of the membranous labyrinth and identify lesions in various parts of the neuraxis as being particularly prone to association with the several semicircular canals. Certainly the neuroanatomist is not able to provide the morphological constituents for the transmission of these several streams of impulses. So far as can be determined, there are no discretely separated secondary vestibular pathways for the conduction of impulses from the semicircular canals in any segregated, distinct fashion. The majority of neurologists also fail to agree with any attempt to localize lesions in the neuraxis as reconcilable with individual canal disturbance.

The central disorders of audition are difficult to recognize. Unilateral lesions in the temporal gyri fail almost uniformly to produce recognizable disturbances in the sense of hearing. Occasional reports of auditory hallucinosis of a relatively unformed kind have been recorded from central lesions, but this situation is in marked and absolute contrast to that presented by visual and olfactory hallucinosis. The auditory hallucinations of the insane are evidently so highly integrated and organized as to be intrapsychic phenomena and probably not in any way connected with the demonstrable local lesions of the central acoustic apparatus.

The associative functions of the temporal cortex are occasionally affected by agenesis or by acquired localized cortical lesions. These disturbances are seen in deaf mutes in the former

instance, tone-deaf or completely amusical individuals and in the latter in the development of word or object deafness, in which the association value of word or sound is lost. These latter disturbances are seen in lesions of the dominant hemisphere in the temporal lobe and adjacent parts of the insula and the parietal lobe.

THE THROAT. The principal purposes of the throat are the ingestion of food and liquid, the inhalation of air, the production of the voice and the protection of the respiratory apparatus.

The throat connected as it is, rostrally with the mouth and caudally with the gastro-intestinal canal, is one of the primitive components of the organism. The primary significance of this structure is for the ingestion of food and water. In the forms developing in an aquatic environment, the needs of the body for oxidation are satisfied by the extraction of oxygen from the water by means of the gills and the discharge by the same means and into the same medium of carbon dioxide and other waste gases. The pulmonary system appears first in those hardy fish and the amphibia which first essayed an invasion of the dry land as a ventral evagination from the gastro-intestinal tract. This development of an offshoot for respiration from a common tube carrying both food and liquid necessitated the elaboration of an intricate mechanism, the larynx, for the separation of solids and liquids from the gases in the interest of protecting the delicate respiratory epithelium from the deleterious activity of liquids and solids. The further evolution of the larynx into an organ capable of producing sound took place concomitantly with the development of the function of hearing.

Since all of these structures and capabilities developed in close contiguity with the head-end of the organism, the requisite nerve supply was supplied by the nearest part of the central nervous system, and these primitive neural connections have been maintained no matter how distantly the peripheral structures have migrated. We therefore find the medulla oblongata supplying the gastro-intestinal tract as far caudally as the ileo-colic junction, the structures of the thorax, and more nearly situated structures anatomically, such as the larynx.

The nerve supply of the throat is supplied by the ninth and tenth nerves if one excludes the special sense of taste, and the very minor contribution to the motor supply of the palate pro-

vided by the facial nerve. The sensory supply of the ninth and tenth cerebral nerves comprises the mucous membrane of the pharynx, the œsophagus, stomach, and small intestine, the larynx, trachea and respiratory epithelium. The motor supply innervates the muscles of the fauces, the pharynx, œsophagus, stomach, small intestine and ileo-cæcal valve in combination and association with the sympathetic system. In addition, the vagus nerve controls the muscles of the larynx, thus contributing the major constituent of phonation.

In addition to the nuclei of the ninth and tenth cerebral nerves, the medulla contains the nuclei of the spinal accessory and hypoglossal nerves which, while they are not intrinsic constituents of the throat, are nevertheless closely associated in function and position.

In the internal organization of the medulla oblongata, certain of the long tracts traversing the neuraxis and certain cerebellar connections are in rather close anatomical relation with the nuclei of the ninth and tenth cerebral nerves. These structures are the spino-thalamic tracts conveying non-discriminative touch sensation and the affective types of sensation, pain and temperature; the mesial fillet transmitting the discriminative types of sensibility and deep sensation; the descending root of the trigeminal nerve subserving the sensory functions of the face, principally pain and temperature, the cerebellar connections controlling coordination and the pyramidal tract providing voluntary motor control. These structures may be affected by lesions which are principally vascular in character, hemorrhage, thrombosis or much more rarely embolism. The blood supply of the medulla oblongata is carried on primarily by numerous small branches of larger trunks. These larger trunks are protected against occlusion by thrombi, etc., by an extensive anastomotic arrangement. The smaller arteries are devoid of anastomotic communications, are in effect end-arteries, and since each one supplies a limited but discrete amount of neural tissue, the occlusion of one or another of these vessels gives rise to relatively localized lesions and a definite train of symptoms and physical signs.

The vagus and hypoglossal nuclei are the principal longitudinal indicators of involvement of the medulla oblongata. Since the vagus nucleus is located somewhat more cephalically than the hypoglossal nucleus, disturbances in vagus innervation indicate a higher level of pathological involvement than do disorders of the lingual musculature. The symptomatic pattern of asso-

ciated neurological disorders resulting from the implication of neighborhood structures indicates the transverse extent and location of the lesion. Lesions may involve the rhombencephalon either in its ventral, its dorsal, or its lateral portions. Certain lesions seem to present a diffuse or spotty incidence and it is only by the careful study of the neural and other functions of the entire individual that a correct interpretation of the actual structures involved can be reached.

There are a number of well-recognized syndromes of the medulla which include disturbance in the innervation of the throat and associated structures. Certain of these syndromes involve a limited number of structures and are relatively easily detected by simple diagnostic procedures. Others involve a large number of structures and present such complicated and variable pictures that schematization is impossible. The neurological disorders dependent upon these intricate involvements are so extensive that their diagnosis may perhaps be left entirely to the neurologist.

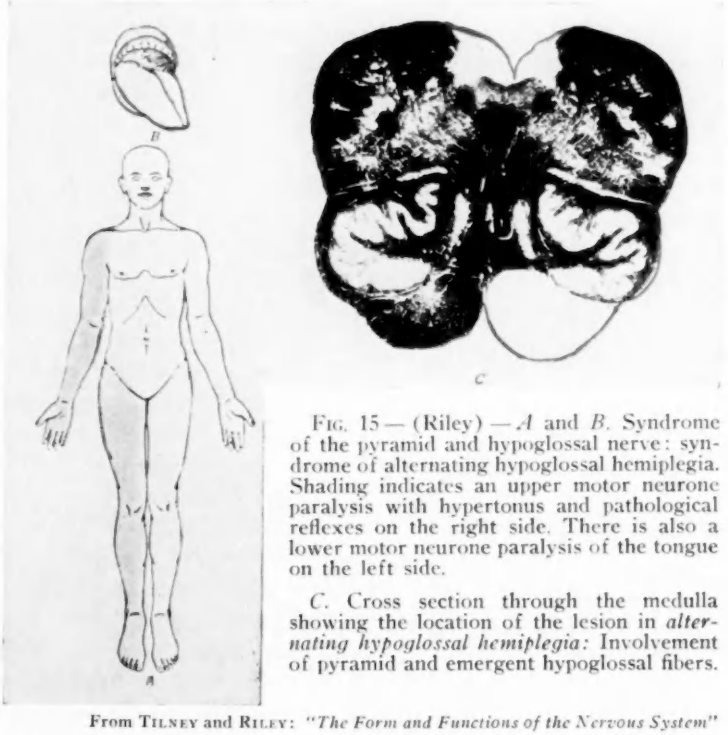

FIG. 15 — (Riley) — *A* and *B*. Syndrome of the pyramid and hypoglossal nerve: syndrome of alternating hypoglossal hemiplegia. Shading indicates an upper motor neurone paralysis with hypertonus and pathological reflexes on the right side. There is also a lower motor neurone paralysis of the tongue on the left side.

C. Cross section through the medulla showing the location of the lesion in ***alternating hypoglossal hemiplegia:*** Involvement of pyramid and emergent hypoglossal fibers.

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If the lesion is dependent upon a single localized vascular, inflammatory or neoplastic process situated on the ventral aspect of the neuraxis involving the pyramidal tract and the emergent fibers of the hypoglossal nerve, the resulting symptom-picture is:

1. *The Syndrome of Alternating Hypoglossol Hemiplegia* (Fig. 15).

The constituent symptoms of this syndrome are:

a. Homolateral
   Paralysis, atrophy and often fibrillation of one-half of the tongue due to direct involvement of the hypoglossal nerve.

b. Contralateral
   Hemiplegia with more or less complete paralysis of the arm and leg, spasticity and increased reflexes due to involvement of the pyramidal tract above its decussation.

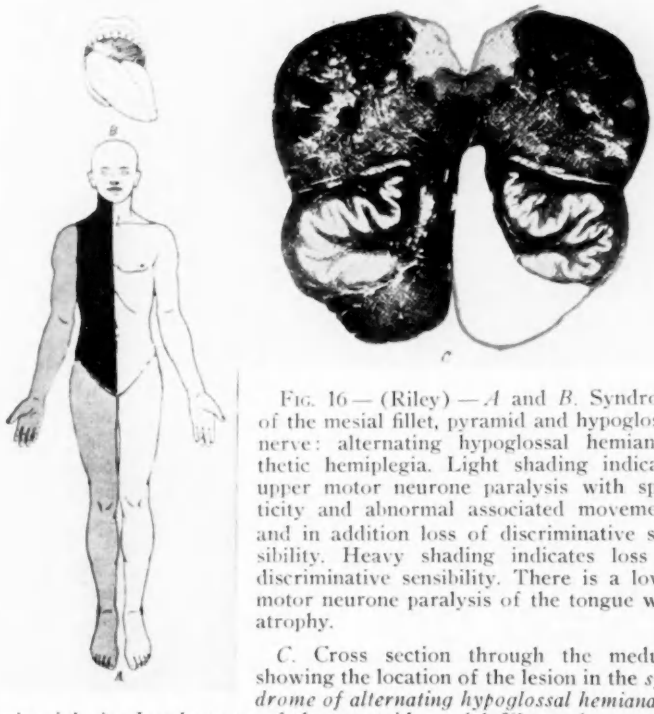

FIG. 16 — (Riley) — *A* and *B*. Syndrome of the mesial fillet, pyramid and hypoglossal nerve: alternating hypoglossal hemianæsthetic hemiplegia. Light shading indicates upper motor neurone paralysis with spasticity and abnormal associated movements and in addition loss of discriminative sensibility. Heavy shading indicates loss of discriminative sensibility. There is a lower motor neurone paralysis of the tongue with atrophy.

C. Cross section through the medulla showing the location of the lesion in the *syndrome of alternating hypoglossal hemianaesthetic hemiplegia:* Involvement of the pyramid, mesial fillet and emergent hypoglossal nerve fibers.

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2. The preceding syndrome may be complicated by the dorsal extension of the lesion to involve the mesial fillet which conveys the discriminative types of sensibility, the sense of position and movement, spatial discrimination, the recognition of the region of the cutaneous surface stimulated and vibratory sensations. This symptom-picture is the *Syndrome of Alternating Hypoglossal Hemianaesthetic Hemiplegia* (Fig. 16).

Isolated circumscribed lesions, principally in the tegmentum of the medulla, may involve some of the smaller nuclei and tracts producing a number of distinguishable syndromes.

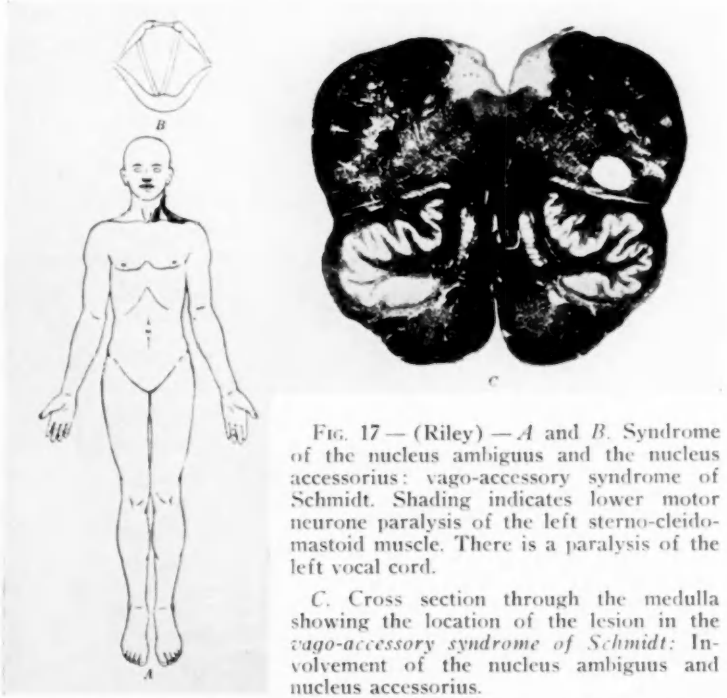

FIG. 17 — (Riley) — *A* and *B*. Syndrome of the nucleus ambiguus and the nucleus accessorius: vago-accessory syndrome of Schmidt. Shading indicates lower motor neurone paralysis of the left sterno-cleido-mastoid muscle. There is a paralysis of the left vocal cord.

C. Cross section through the medulla showing the location of the lesion in the *vago-accessory syndrome of Schmidt:* Involvement of the nucleus ambiguus and nucleus accessorius.

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3. *The Vago-Accessory Syndrome of Schmidt* (Fig. 17).

The constituent symptoms of this syndrome are:

Homolateral

a. Partial paralysis of the sterno-cleido-mastoid and trapezius muscles, resulting in inability to turn the head away from the side of the lesion and a deviation of the head toward the

side of the lesion due to paralysis of one cephalogyric group by reason of a nuclear lesion affecting the spinal accessory nerve of the unopposed activity of the unparalyzed group of muscles.

b. Paralysis of the vocal cord by reason of involvement of the ventral vagus motor nucleus, the nucleus ambiguus.

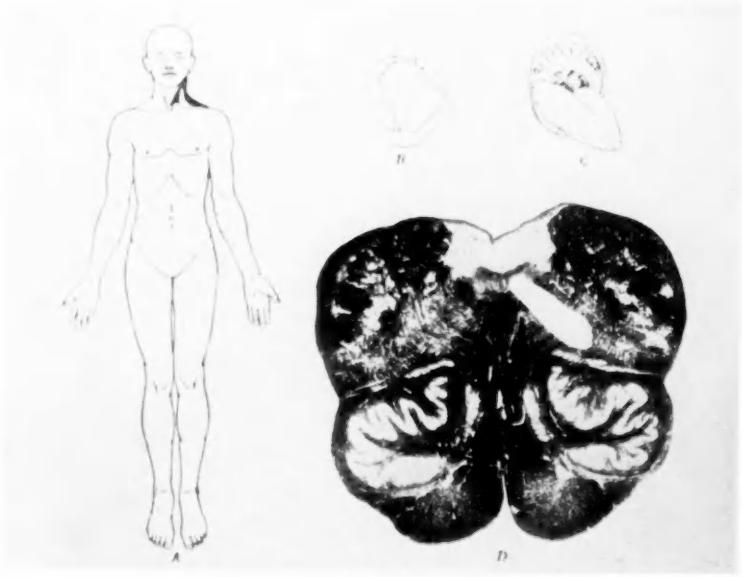

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FIG. 18 — (Riley) — *A*, *B* and *C*. Syndrome of the nucleus ambiguus, nucleus accessorius and nucleus hypoglossus; syndrome of vago-accessory hypoglossal paralysis: syndrome of Jackson. Shaded area indicates a lower motor neurone paralysis of the left sterno-cleido-mastoid and trapezius muscles. There is also a paralysis of the left vocal cord, the left half of the tongue and the left half of the soft palate.

D. Cross section of medulla showing involvement of nucleus hypoglossus and nucleus ambiguus.

4. *The Syndrome of Vago-accessory-hypoglossal Paralysis: the Syndrome of Jackson* (Fig. 18).

The constituent symptoms of this syndrome are:

Homolateral

a. Paralysis and atrophy of one-half of the tongue due to involvement of the hypoglossal nucleus.

b. Partial paralysis of cephalogyric movement away from the affected side as a result of implication of the spinal accessory nucleus.
c. Paralysis of one vocal cord due to involvement of the ventral vagus motor nucleus.
d. Paralysis of one-half of the soft palate, due to the same cause.

5. *The Syndrome of the Nucleus Ambiguus and the Spino-thalamic Tract: the Syndrome of Avellis* (Fig. 19).

The constituent symptoms of this syndrome are:

a. Homolateral
Paralysis of one-half of the uvula and one vocal cord due to involvement of the nucleus ambiguus.
b. Contralateral
Loss of the ability to recognize sensations of pain, heat and cold due to involvement of the spino-thalamic tract supplying the opposite half of the body.

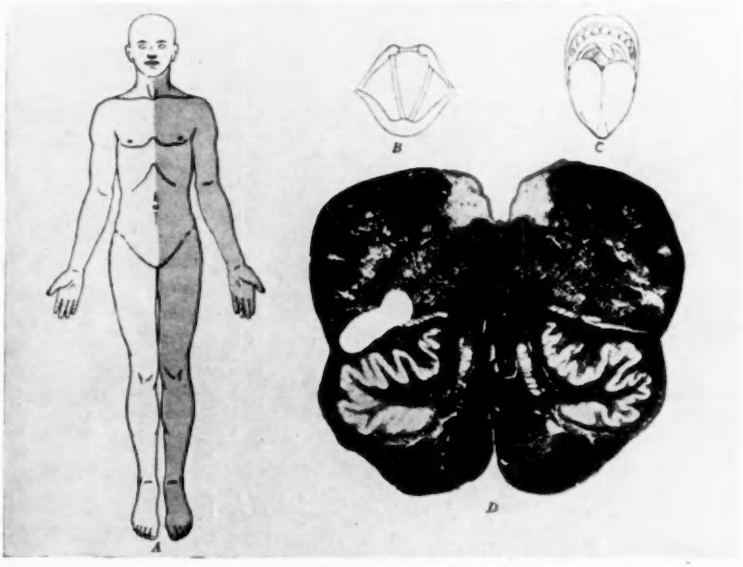

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FIG. 19 — (Riley) — *A*, *B* and *C*. Syndrome of the nucleus ambiguus and spinal fillet (spino-thalamic tract); syndrome of Avellis. Shading indicates a loss of pain and temperature sensibility. There is a lower neurone paralysis of the right vocal cord and right half of the soft palate.

D. Cross section through the medulla showing the location of the lesion in the *syndrome of Avellis:* Involvement of the nucleus ambiguus and spino-thalamic tract (spinal fillet).

The more extensive syndromes of the bulb implicate a greater number of structures and therefore have a much richer symptomatic picture than the preceding syndromes.

6. *The Syndrome of the Posterior Inferior Cerebellar Artery* (Fig. 20).

The constituent symptoms of this syndrome are:

a. Homolateral

(1) Paralysis of one-half of the uvula and one vocal cord due to a lesion of the nucleus ambiguus.

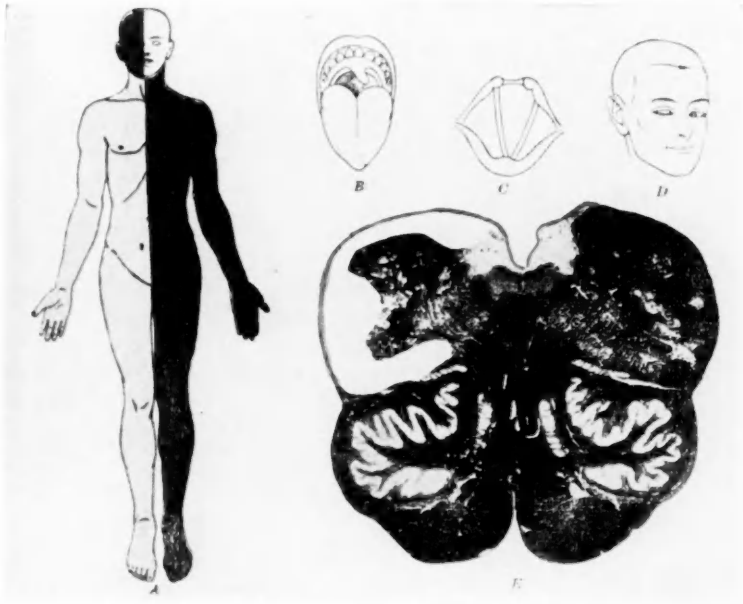

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FIG. 20 — (Riley) — *A, B, C* and *D*. Syndrome of the circumferential and intermediate zones; syndrome of the posterior inferior cerebellar artery. Light shading indicates a hemiasynergia, swaying and lateropulsion to the right. Shading in face indicates a loss of all types of sensibility. Heavy shading indicates a loss of or decrease in pain and temperature sensibility. There is a lower motor neurone paralysis of the right half of the soft palate and the right vocal cord with a right conjugate deviation of the eyes.

E. Cross section through the medulla showing the location of the lesion in the *syndrome of the posterior inferior cerebellar artery:* Involvement of the spino-cerebellar, spino-thalamic and descending trigeminal tracts, together with the nucleus ambiguus and vestibular nuclei.

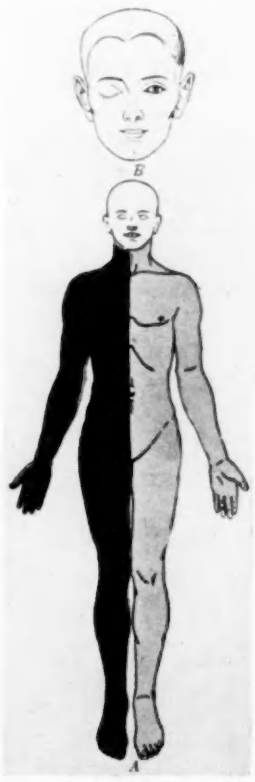
(2) Loss of sensation, principally pain and temperature, on the side of the face due to an involvement of the descending root of the trigeminal nerve.

(3) Hemiasynergia and incoordination due to involvement of the inferior cerebellar peduncle.

b. Contralateral

(1) Paralysis of conjugate gaze away from the affected side, the result of the involvement of the lateral gaze pyramidal tract bundle before its crossing and the unapposed action of the other oculogyric control. This involvement must be at a higher level than indicated in the diagram.

(2) Loss of pain and temperature sensibility due to involvement of the spino-thalamic tract which conveys these types of sensation.



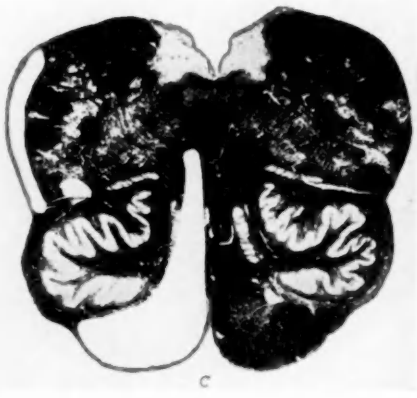


FIG. 21 — (Riley) — *A* and *B*. Syndrome due to multiple lesions in the medulla oblongata: syndrome of Babinski-Nageotte. Light shading indicates an upper motor neurone paralysis and also a hemianæsthesia involving descriminative sensibility. Heavy shading indicates hemiasynergia, hemiataxia and lateropulsion to the right. There is also myosis, enophthalmos and ptosis on the right side.

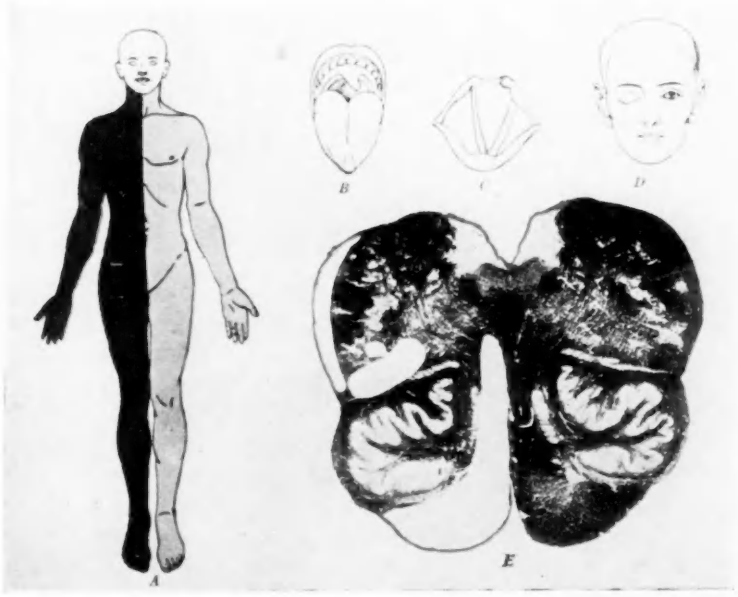
C. Cross section through the medulla showing the location of the lesions in the *syndrome of Babinski-Nageotte:* Involvement of the pyramid, mesial fillet, spino-cerebellar tracts and oculo-pupillary nucleus.

From TILNEY and RILEY: *"The Form and Functions of the Nervous System"*

Multiple lesions within the bulb may present almost any combination of the preceding symptom-pictures and the definite determination of the actual structures involved can only be made by careful examination and evaluation of all of the evidence of disturbed function which can be elicited by a complete neurological examination.

Certain other involvements of the neural control over the muscles of the fauces, the pharynx, larynx and tongue may result from unilateral or bilateral involvement of the pyramidal tract and its aberrant fasciculi which supply the nuclei of the cerebral nerves controlling these structures.

As one sees paralyses of conjugate gaze resulting from supranuclear lesions of the oculomotor pathways, so one may meet



From TILNEY and RILEY: *"The Form and Functions of the Nervous System"*

FIG. 22 — (Riley) — *A, B, C* and *D*. Syndrome of multiple lesions in the medulla oblongata: syndrome of Cestan-Chenais. Light shading indicates an upper motor neurone paralysis and a hemianæsthesia affecting all types of sensibility. Heavy shading indicates a hemiasynergia, hemiataxia and lateropulsion to the right. There is a lower motor neurone paralysis of the right half of the soft palate and left vocal cord. There is also myosis, enophthalmos and ptosis on the right side.

E. Cross section through the medulla showing the location of the multiple lesions in the *syndrome of Cestan-Chenais:* Involvement of the pyramid, mesial fillet, spino-thalamic and spino-cerebellar tracts with the nucleus ambiguus and nucleus oculo-pupillaris.

with similar disturbances in the mouth, larynx, and pharynx. A hemiplegia may be limited to the cerebral nerves or it may involve also the extremities or only the latter structures may be involved, according to the part of the pyramidal tract involved. Unilateral, so-called upper motor neurone paralysis may rarely involve the uvula, pharynx, or tongue. Under these circumstances, no rapid atrophy ensues and the reflexes of the throat are still active. Such lesions may, if repeated, involve first one and then the other side, resulting in the pseudo-bulbar palsy with its disturbances in the innervation of palate, pharynx, larynx and tongue with a resulting loss of voluntary control and the development of the typical so-called pseudo-bulbar speech. The reflexes are still present, however, due to the fact that the nuclei of the cerebral nerves are not involved and the reflex-arcs are undisturbed. Only voluntary control is affected. In the true bulbar palsy, which is a polioencephalitis, the affection is one of the nuclei itself and therefore is accompanied by atrophy and loss of reflex activity. This is a much more dangerous condition, since the protective mechanism for the larynx and the respiratory system is put out of function and inhalation pneumonia usually closes the picture.

The possibilities for involvement of the cerebral nerves supplying the eye, ear, nose and throat by the various ills to which man is heir are almost limitless. Localized lesions such as neoplasms, aneurysms, vascular lesions, cysts or injury can usually be made to fit into some more or less clearly defined syndrome such as those outlined above. Diffuse or generalized infectious processes, multiple vascular lesions or injuries may produce any bizarre or uncorrelated grouping of neural involvements. These are impossible of schematization, since any pattern may be developed depending upon the actual localization of the lesions, and the attendant implication of neural structure. An appreciation of the anatomical arrangements and relations, however, will indicate the true solution of these intricate problems and provide a correct diagnosis.

There is no royal road to the unravelling of these complicated problems and only by the exact knowledge of the anatomy of the central and peripheral nervous systems and the components of these systems together with a true appreciation of the type of the disorder affecting the individual can the proper answer be provided.

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THE TREATMENT OF ALLERGIC RHINITIS WITH PARTICULAR REFERENCE TO IONIZATION AND THE CONTROL OF CHRONIC VASOMOTOR RHINITIS*

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SAINT LOUIS

Twenty months ago the Department of Otolaryngology in the Medical School of Washington University decided to make a study of allergic rhinitis. The late Frank E. Ball of Muncie, Indiana, created the Frank E. Ball Research Fund to carry out this study.

Antedating this research two observations regarding ionization had already been made in the Department. McMahon (Annals of Otology, Rhinology and Laryngology, 44:643, 1934) reported that ionization in dogs produced a fibrosis of the subepithelial tissue with marked hyperplastic bone changes; that the epithelial changes in the nose consisted of a primary destruction of cells with a later return to normal. Alexander and Alexander (Journal of Allergy, 6:240, 1935) reported that it was the patient suffering from hay fever and had no reagins in the blood stream who was more likely to get a good result from ionization while the one who had an abundance of reagins in the blood stream as a rule had an unsatisfactory result following the treatment. Hansel (Allergy of Nose and Paranasal Sinuses, Mosby and Company) made a most excellent and comprehensive review of allergic rhinitis.

A group of scientists, some doctors of medicine and some not, were engaged to further this investigation. This is a report of part of the work done. The immunological work reported was done by L. D. Linton; the pathological work was done by and

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the study of the action of radium and of surgical diathermy on the mucous membrane was made by L. W. Dean, Jr.; the study of the action of alcohol and of carbolic acid in the nasal mucous membranes of animals was made by Charles Mahoney. The study of the action of trichloracetic acid and actual cautery on the nasal mucous membranes by H. M. Smit. Wenner (Annals of Otology, Rhinology and Laryngology, 45:31, 1936) has shown that ionization of the frog's foot slowed the circulation in from eight to fifteen minutes; that a clumping of the red blood cells took place in 45 minutes; that the circulation was completely stopped in one hour; that if the electric current alone was used the circulation was not stopped and the clumping of blood cells did not take place. Similar observations were made on a rabbit's ear. Wenner studied the influence of zinc iontophoresis on the feet of guinea pigs and concluded that the resulting ulceration of the skin and gangrene of the feet was probably in part, at least, caused by the circulatory disturbances and made the observation that small electrodes if used in ionization were more likely to cause ulceration than the larger ones.

Later Wenner (Annals of Otology, Rhinology and Laryngology, 45:31, 1936) demonstrated that so-called ionization was in reality iontophoresis, that the zinc ions actually penetrated the skin and cartilage of the rabbits' ears during the process of ionization.

Linton (Annals of Otology, Rhinology and Laryngology, 45: 63, 1936) made intramucosal tests before and after iontophoresis. If the patient gave a positive intramucosal reaction before ionization he always gave a positive reaction after the treatment. Some patients who received relief from allergic rhinitis from the use of iontophoresis did not react to the topical application of allergens to the mucosa, but they always reacted to the intramucosal injection of the ragweed pollen if they were ragweed hay fever cases. He secured a reaction in one case as early as eleven days after iontophoresis. He has since noted that trauma and other procedures in the nose influence the reaction of the mucous membrane to allergens; that is, for instance the removal of polyps, submucous resection, etc., may interfere with the reactions when intramucosal tests are made. He has also noted that local desensitization of the mucous membrane may result from the injection of an allergen. For example, in a case of hay fever that has been ionized an intramucosal injection of ragweed pollen was given to test the sensitivity of the mucous membrane.

This resulted in a terrific attack of hay fever which could only be controlled by an injection of adrenalin. A repetition of this injection ten days later produced no reaction. It has been noted many times that intramucosal tests in allergic rhinitis may themselves produce a clinical improvement.

H. M. Smit (Annals of Otology, Rhinology and Laryngology, 45:38, 1936) studied the tissue changes following zinc iontophoresis. His findings are as follows: "Summarizing the tissue changes which one finds following zinc iontophoresis there occur: coagulative necrosis of the epithelium; early stages of inflammation with polymorphonuclear leucocytes which are later replaced by round cells; atrophy of glands and loss of glands; metaplastic changes in glands and ducts; formation of stratified squamous epithelium, which later returns to normal, ciliated columnar (if there is not a chronic inflammatory process present); fibrosis of tunica propria; dehydration of edematous tissue; fibrosis of blood vessels and in some cases cartilaginous necrosis."

Boling (Archives of Otolaryngology, December 1935) working in the Department of Cytology at Washington University showed that following ionization of the sheep's nose there was a complete regeneration of the ciliated epithelium. Wenner has made the same observation on cats. In the human subject, probably because of the fact that we were dealing with chronic inflammation of the mucous membrane in the beginning, we have always found areas of persistent squamous cells following the ionization. Biopsies were made from the same area on the inferior turbinate before and after ionization. Before ionization the cells were all ciliated. One month after ionization no ciliated epithelium was present, the epithelium being all stratified squamous. Three months after ionization the only ciliated epithelium found is in a small depression in the mucous membrane which may have caused the epithelium at that point to be unaffected by ionization. It is true that the more insult there is to the nasal mucous membrane the more stratified epithelium will be found. Theoretically, it is to be expected that following the necrosis of the superficial tissue that we get with ionization there will be more squamous epithelium than before.

During the last year we have been interested in the activity of the cilia of the cells that were regenerated following ionization. One patient had particularly good cilia in the microscopic section of a biopsy after ionization. Some of these cells were removed and studied by Miss Pfingsten. She compared their activity with

the activity of the cilia of normal cells and also the length of time that the cilia beat when kept in nutrient solution in the culture chamber following removal. The activity and the length of time that they beat were practically the same in the normal and in the regenerated ciliated cells. This patient had been ionized some eight or ten months before because of a chronic vasomotor rhinitis. The mucous membrane of this patient was much drier than normal. Several members of the staff looked at this patient's nose with me and we all agreed that while the cilia might be beating in the lower layer of mucus, that the mucus covering the membrane was so dry that it was impossible for the cilia to produce any movement on the surface.

We have convinced ourselves (L. W. Dean, C. S. Linton, L. D. Linton: Annals of Otology, Rhinology and Laryngology, 44:317, 1935) that some form of mucosal test is necessary in studying allergic rhinitis. The comparison of the results of intramucosal tests with skin tests shows that often the skin will give a negative reaction for an allergen to which the mucous membrane of the nose is sensitive. (Linton: Archives of Otolaryngology, 23:679, June 1936). It has been found that by avoiding the allergens to which the mucous membrane is sensitive and to which the skin did not react a satisfactory result has been secured.

During the last year Wenner and Alexander (In press, Report to appear in the Archives of Otolaryngology) have made a study of the action of ionization and of the galvanic current on the vasomotor mechanism of the nose. They proved that the blood vessels of the nasal mucous membrane failed to react to vasoconstrictors and vasodilators for six weeks after zinc ionization in the cat's nose and for four weeks after treatment with the galvanic current. They were unable to determine whether this was due to a paralysis of the vasomotor nerves or loss of vascular tone. The absorption of drugs by the mucosa was not affected by ionization.

During the last year we have also been interested in the influence of ionization in the nasal mucous membrane upon the absorption of allergens by the membrane. Three patients who were ionized because of chronic vasomotor rhinitis were studied before and after ionization in order to determine the relative absorption of allergens through the untreated and treated mucous membrane. The method of determining the absorption of allergens through the mucous membrane of the nose described by Sulzberger and Vaughan (Journal of Allergy, 5:554, 1934) was used. These

three patients were non-allergic to ragweed. Blood containing reagins to ragweed was injected into the skin of one arm. This produced a local sensitivity to ragweed pollen. A similar amount of blood without the reagins to ragweed was injected into the other arm. In the first case tested, the patient had had one side of the nose ionized sixteen days before. Twenty-four hours after injecting the arm the ragweed pollen was placed in the un-ionized side of the nose. In fifteen minutes there was a reaction apparent on the arm at the site of the injection of the blood containing the ragweed reagin. The other arm did not react. This was repeated on the ionized side of the nose. There was no reaction on either arm in twenty-four hours. The other two patients had the same test made except that the test was made before and after ionization. In the first case before ionization the reaction in the arm started in ten minutes and reached a maximum in thirty-three minutes. Eight days following the ionization the test was repeated with negative results. In the second case before ionization the reaction started in thirteen minutes after the injection and reached its maximum in one hour and thirty minutes. Twenty-one days after the ionization the absorption test was again made. The reaction in the arm in this instance started in forty-three minutes and reached its maximum in one hour and forty-three minutes. The reaction following the ionization was more marked than that which preceded it.

We have only had the opportunity to study the influence of ionization on the absorption of allergens by the nasal mucosa in the three cases. It is interesting at least to note that in two cases there was no evidence of absorption at all of the ragweed pollen through the mucous membrane of the ionized nose while in the third case the absorption was delayed.

Further studies during the last year on the human nose, taking biopsies before and after ionization have suggested that the final results of ionization were: fibrosis and thinning of the tunica propria, the basement membrane is thicker and more dense; there is a hyperplasia of the epithelium with increase in metaplasia; very few goblet cells are present; sometimes there is a thickening of the vessel walls particularly the capillaries; the number of eosinophils in the tissue is not decreased; the secretory glands are partially destroyed.

Some of the acini of glands appear to be completely obliterated by cellular hyperplasia. In others the secretory cells are atrophied. Marked inflammation may be present around the acini.

These changes result in an interference with the secretory function of the nasal membrane and the membrane does not swell as easily as it did before. Biopsies taken from the human nose are necessarily superficial. Periosteal changes have been found in the occasional biopsy where a piece of bone was secured. In the human biopsies the cartilage of the septum could not be studied. In dogs and guinea pigs some cartilage cells were killed by the ionization.

We have proved to our satisfaction that ionization does not change the allergic status of the body in any way. While dusting allergens in the nose may not cause a reaction after ionization the tissues of the nose always react as before ionization to the intramucosal injections of allergens. With intramucosal tests there is a difference; namely, the tissues do not swell as before. There is no change in the reagin content of the blood. It has been found, however, that by using different individuals to test the reagin content of the blood, using the same blood different results may be secured. This may explain the suggestion that has been made that the allergic status of the individual is changed by the ionization.

We do not find it necessary to use ionization for the control of hay fever. It is used to control the chronic vasomotro rhinitis which persists after the hay fever season. It is interesting to note that we had on September 1, 1936, in our research clinic five patients who were ionized one or two years ago for hay fever with unsatisfactory results. One of these patients is doing very well with desensitization, diet, hormones, etc. The other four are not doing well. The patients who are highly sensitive to ragweed pollen, those who have +4 reagins in the blood stream, are very difficult to handle with any kind of treatment except the avoidance of allergens. All other hay fever patients in the clinic are doing well with desensitization, diet, avoiding other allergens to which they are sensitive, correction of endocrine dyscrasias, etc.

We have used ionization for the control of chronic vasomotor rhinitis when other forms of treatment have failed. It has been used when all the mucous membrane of the nose, that on the septum and in the meati, as well as on the turbinates, is boggy.

In treating allergic rhinitis an exact diagnosis is essential. I have seen patients with edematous nasal mucous membranes where at first glance I have thought it was allergic rhinitis. Some of these cases after being studied for a year and a half have been

proved not to be allergic. The only thing that brought about an improvement in the nasal condition was a proper diet plus cortin.

Rackemann states that 50 per cent of the cases of vasomotor rhinitis are non-allergic (Clinical Allergy, Asthma and Hay Fever, the Macmillan Co., 1931). Cameron of Glasgow (Journal of Laryngology and Otology, London, Vol. 50:493, July 1935) puts it at 40 per cent. These percentages, as I view it, are very high. No one has ever proven that the cases of non-allergic vasomotor rhinitis were not at one time allergic. We had under observation a patient with allergic rhinitis who secured a most excellent result by the avoidance of certain allergens to which she was sensitive and by undergoing a submucous operation. Examination of the nose showed good breathing space; the turbinates, however, were paler and softer than normal. The biopsy of the tissue taken after the treatment showed edematous tissue with no increase of eosinophils in the tissue.

Vasomotor disturbances in the nose may result from infection and inflammation. Even a simple irritation of the mucous membrane causes vasomotor changes. Clinically we must assume that there are many cases of vasomotor rhinitis which are not at the time of the examination allergic. Further we must realize that many of these conditions will improve with treatment for dietary and endocrine defects.

Repeated attacks of acute allergic rhinitis leave behind a chronic vasomotor rhinitis. During the last year I have seen only one hay fever patient that had a normal mucous membrane. This patient had had only one attack of hay fever. The treatment of allergic rhinitis is so intimately tied up with the treatment of chronic vasomotor rhinitis that the two cannot be separated. I feel that every case of edematous nasal polyps and every case of chronic vasomotor rhinitis should have a complete allergic work-up. We yet have not seen a case of edema of the mucous membrane with a great increase of eosinophils in the nasal discharge that did not prove to be allergic. On the other hand we have all seen cases where with one or two or three studies of the nasal discharge no increase of eosinophils has been found, but further study of the vasomotor rhinitis proved the case to be allergic and a good result was secured by allergic treatment.

Every case of boggy mucous membrane should not only have a complete allergic study but should have dietary, bacteriological and endocrine studies made. The first thing to do in the treatment of all cases of chronic vasomotor rhinitis is to correct the allergic

condition if it is present. Dietary defects and endocrine disturbances should be corrected and infection should be eradicated. If this has been done there will be a certain percentage of boggy noses where a good result can be secured only by some type of therapy applied to the mucous membrane of the nose.

One of the essential things in the treatment of allergic rhinitis is that the patient be under the care of a rhinologist. The examination of the nose is necessary for a proper diagnosis. It is only by watching the nose that one gets the best idea as to the result of a given therapeutic procedure. Last, and by no means least, is the fact that one of the essential things in the treatment of allergic rhinitis is to bring the nose into as normal a condition as possible. Infection of the nasal sinuses must be corrected. In perennial allergy we often get good results following the correction of deflected septa, the removal of exostoses or after the removal of adenoids. Before we knew very much about allergy we knew that cases of vasomotor rhinitis were often favorably influenced by the removal of adenoids in children. Most striking results often follow the correction of nasal lesions.

Patients with allergic rhinitis where the biopsy has shown a typical allergic picture have secured complete subjective relief with objective shrinking of the mucous membrane of the nose following a submucous operation or the removal of adenoids. One might immediately ask the question, "How long will the patient be benefited?" The question might well be answered by asking, "How long is a patient with allergic rhinitis benefited by desensitization or any other form of treatment?" It is often necessary to eradicate a chronic vasomotor rhinitis in order to get the nose normal. In certain cases of chronic vasomotor rhinitis ionization may be used. Its best and most permanent result is secured in chronic non-allergic vasomotor rhinitis. I have observed a patient with chronic vasomotor rhinitis that had one side of his nose ionized. Three months before the patient had had acute upper respiratory upsets which were non-allergic. The un-ionized side of the nose was pink, blocked and actively secreting. The ionized side of the nose was dry with good breathing space and the mucous membrane, instead of being pink and actively secreting, was pale and only slightly moist.

A second essential in the treatment of allergic rhinitis is a most careful study of the patient. With our patients we are definitely of the opinion that, if after careful study we are not getting a good result from treatment, we have overlooked something and

the patient must be studied again. It is a common thing in the management of the treatment of perennial cases of allergy for us to go back and restudy the case.

The history is a most important part of the study and still one must be sure not to be too much influenced by it. The finding of a gastro-intestinal allergy does not make a diagnosis of an allergic rhinitis in the patient if he has an upper respiratory upset. A patient may have a gastro-intestinal allergy and still have a chronic vasomotor rhinitis which is not necessarily allergic.

Nasal smears and a blood count should be made in every case. It is certain that in trichinosis and other parasitic diseases and in digitalis and other forms of poisoning one may have a great increase in the eosinophils in the nasal discharge without having an allergic rhinitis. At the same time there will be an increase in the eosinophils of the blood. Up to this time I have never seen a patient with an edematous nose and a marked increase in the eosinophils in the nasal discharge that did not prove to be allergic. An exception to this may be found at any time. An x-ray examination of the sinuses should be made because a thickened sinus lining is always suggestive of an allergic condition.

A bacteriological examination is most important whether there is definite infection of the nasal sinuses or not. There is such a thing as bacterial allergy. Biopsies of the tissue present the same picture of edema with a great increase in eosinophils as is found in other forms of allergic rhinitis. If there is any suspicion of bacterial allergy and organisms have been cultured which are suitable for making a vaccine the tests must wait the preparation of a vaccine. Skin reactions and systemic reactions follow the use of vaccine in cases of bacterial allergy. We are inclined to think the greater the reaction the better the prognosis from the use of a vaccine.

Dietary deficiencies must be sought for. We should not put the patient on a diet that is made deficient by excluding from the diet certain substances to which the patient is sensitive. It is inexcusable to take from the food supply a given essential food without replacing it with something equally good. This is sometimes a definite problem.

We have proven conclusively in our clinic that we do not get as good results from the administration of vaccines or from the desensitization of patients with allergic rhinitis if the patient is not on a proper diet. Ragweed hay fever patients are sensi-

tive to other substances, particularly foods. Not only are these patients improved by the avoidance of foods and other allergens to which they are sensitive, but a good diet helps with the hay fever.

Trauma of the nasal mucous membrane of the nose and malnutrition both do something to the protective lining of the nose that permits allergens to enter into the tissue. This is the reason why we have so many cases of asthma following operations on the tonsils. It is interesting to note that some of these patients have only asthma when they return to the community where the tonsils were removed. The break in the protective epithelium permits the entrance of allergens into the tissue and sensitization results.

Endocrine disturbances should be sought for and, if present, corrected. We have a basal rate determination made in every case of allergic rhinitis. We make skin tests in every case. We realize that they are often more an indication of the past allergic history of the patient than the present. Reagins are formed in all of the tissues of the body except the cerebro-spinal fluid and the aqueous humor of the eye. As soon as the acute allergic attack is over the organism starts excreting the reagins. They disappear from the blood and other circulating media of the body first. The skin is one of the structures from which they disappear last. A child may have been sensitive to milk at the age of two years and the sensitivity may have disappeared before it reaches two and one-half years of age and still give a positive skin reaction to milk at the age of fifteen years. We would be unwilling to treat cases of allergic rhinitis without some test of the sensitivity of the mucous membrane of the nose to the various suspected substances. We like the intramucosal test. We have done more than a thousand tests without any deleterious results. It is axiomatic that we have not used sera and other similar substances which are very likely to cause severe systemic reactions. There is little or no danger with the various pollens and foods in the proper dilutions. Certainly it is a common thing to find the mucous membrane of the nose sensitive to a substance to which the skin does not react.

Passive transfers are not done as a routine procedure. They are done whenever we wish to know just how sensitive a given patient is to a certain allergen. Before making a passive transfer careful serological tests are made. The serum of the patient to be tested is injected into the skin of a non-allergic person. If

reagins to ragweed are in the serum used they produce local sensitization at the site injected. At the end of three hours the site of the injection is tested with ragweed pollen and the amount of reaction determines the amount of reagin or antibodies to those allergens in the serum.

The leukopenic index as worked out by Gay has proved to be of the greatest value to us in studying food allergy. The technician doing this work must be capable of making exact blood counts. It takes much of her time. In a number of cases, after a thorough study, if the treatment is not satisfactory and we do not get a good result, the leukopenic index to determine food sensitivity is used. The results secured by this procedure, particularly where clinical symptoms are produced, are exact guides for therapeutic purposes. We are aided in the study of our case by the results secured by the withholding of certain allergens or by the symptoms produced by their administration.

After the completion of our study of the case, the treatment is the avoidance of allergens, desensitization, dietary measures, making the nose normal, and, if it is indicated, endocrine therapy.

In connection with desensitization it is well to remember that if a person is made sensitive to one protein he may react to proteins with similar composition. In the same way in desensitization using an allergen may desensitize the patient to other allergens with a similar chemical composition.

Getting rid of the chronic boggy edematous nasal membrane which so often complicates allergic rhinitis is a very difficult thing. As we said before, ionization often gives good results. Hollander (Archives of Physical Therapy, X-Ray and Radium, 15:587, 1934) has used ionization for ten years with good results in intumescent rhinitis.

Ionization may give a good result in patients with a maximum of chronic vasomotor rhinitis and a minimum of allergy. A patient suffered all the year around from nasal stoppage and sneezing. During the hay fever season she was confined to her bed. She worked in an atmosphere containing white flour dust. White flour gave a mild reaction in the nose; there were no reagins for white flour in her blood. The mucous membrane reacted violently to ragweed pollen. She had +4 reagins for ragweed in her blood. Ionization controlled her vasomotor rhinitis and gave her an excellent result until the next hay fever season when she had as much trouble as ever. After the hay fever there was a partial return of her chronic vasomotor rhinitis.

Among other things the following have been recommended for the control of chronic vasomotor rhinitis; radium, cautery, surgical diathermy, 95% alcohol, and concentrated carbolic acid and 50% trichloracetic acid. A study was made of the action of each of these on the tissues with the hope that something as efficient as ionization with a less deleterious action may be found. All of the things mentioned that are said to be beneficial in chronic vasomotor rhinitis act by splinting the membrane. The resulting fibrosis keeps the membrane from swelling.

A study was made of the use of radium in the treatment of nasal polyps and its effect on the nasal mucosa. Several authors have been prompted to use radium in the treatment of nasal polyps because of the fact that radium stimulates the formation of fibrous tissue. It has been a common observation that the more fibrous a polyp is found to be, the less are its chances of recurring after removal.

The first to use radium for this purpose was Sluder (Laryngoscope, 34:124, 1924). Others who used this method and modified it in various ways were Lyons (Ann. of Otol., Rhin. and Laryng., 15:249, 1931), McCullagh and Robinson (Arch. of Otolaryn., 4:215, 1926), Robinson (Arch. of Otolaryn., 10:72, 1929), and Scal (Laryngoscope, 37:735, 1927, and Arch. of Otolaryn., 16: 199, 1932). The general method used by these authors was to remove the polyps first. Then, in 24 to 48 hours, to place radium in the nose, using 150 to 400 milligram hours. Lyons used 400 milligram hours three times (48 hours after, 8 days after and 15 days after the removal of the polyps) without noting any burning or other harmful effects. The above authors all found a delay in the recurrence of polyps and all stress treatment of the allergic condition or infection as the only means of complete cure. No detailed microscopic findings were reported by these observers.

In order to study the changes brought about in edematous polyps by radium we placed 50 milligrams of radium in each side of the nose of a patient, leaving it in place for four hours (total dose 200 milligram hours on each side). The polys became diffusely reddened in three days and there was a profuse mucopurulent discharge. The edema of the polyps and adjacent mucosa was increased. This lasted for two weeks, at the end of which time the polyps were again pale and the same size as before treatment.

Polyps removed before radium treatment were covered by pseudostratified columnar ciliated epithelium lying on a thick

pale basement membrane. The stroma was very edematous and scattered through it were a few lymphocytes, plasma cells and eosinophils.

One week after radium treatment the epithelium was unchanged. The small blood vessels were engorged. In the spaces between the fibers of the reticulum in the stroma was an increased amount of fluid which stained more deeply with eosin. A much greater number of plasma cells and lymphocytes were found in the stroma. The number of eosinophils was unchanged.

Three weeks after radium treatment changes in the epithelium appeared. These cells lost their cilia, the nuclei became pycnotic, the cytoplasm granular and numerous vacuoles appeared. The pseudostratified epithelium was reduced to a single layer of cuboidal cells. In the stroma were young fibroblasts in numbers sufficient to change its appearance entirely. Edema was still present, the fluid in tissue spaces now taking no stain. Plasma cells and lymphocytes were present in large numbers, the eosinophils remaining in the same numbers as before.

Thus we have definite evidence of a stimulation of fibrous hyperplasia in edematous polyps occurring two weeks after radiation. In addition there is temporary edema and congestion, an increase in the chronic inflammatory cells infiltrating the stroma and destructive cytological changes in the epithelium.

In order to study the end results of the action of radium on the nasal mucosa, 400 milligram hours of radium was used in one side of a dog's nose. In six weeks the dog was killed and serial sections made of the snout.

At this point should be mentioned the work of Bernheimer and Cutler (Arch. of Otolaryn., 16:561, 1932, and Arch. of Otolaryng., 17:658, 1933) and Bernheimer (Arch. of Otolaryn., 22:165, 1935). They studied the action of radium on the allergic nasal mucosa of humans and give detailed microscopic findings and illustrations. The end result was one of fibrosis and loss of edema with no evidence of atrophic or infectious changes in the mucosa.

The only change noted in the dog at six weeks was fibrosis on the treated side. The tunica propria was the same thickness on both sides but more dense on the treated side. Glands, epithelium, periosteum and vessels remain unchanged although there may have been temporary changes present earlier.

A study was also made of the microscopic changes brought about by the use of the submucous diathermy. The subject of

this study was a 27 year old woman suffering from sneezing and rhinorrhœa on an allergic basis. Relief was not obtained by the withdrawal of allergens.

Before diathermy biopsies of the inferior turbinates showed a mucosa covered with pseudostratified ciliated columnar epithelium with many goblet cells. There were many areas of edema in the tunica propria, especially in the more superficial layers. A dense infiltration of lymphocytes, plasma cells and eosinophils was present. The glands were increased in number.

One month after diathermy biopsy of the inferior turbinate showed the epithelium to be unchanged. The basement membrane was thicker but less dense than formerly. The inflammatory infiltration was unchanged. In the tunica propria was a dense band of fibrous tissue. Its fibers radiated inward to the fibrous layer of the periosteum and out to the epithelial layer. The epithelium was drawn in forming a deep fissure on the surface of the turbinate as though by contraction of the scar tissue. The glands in this region were distorted by the scarring.

At the time of biopsy, one month after diathermy, diathermy was repeated on the left side because some edema persisted. A week later another biopsy was taken. The same changes were observed, that is, scarring and loss of edema. In addition there was a metaplasia of the glandular epithelium and the epithelium of the ducts, whole groups of acini being lined by stratified epithelium.

In London in 1897 it was said that 50 per cent trichloracetic acid was a good thing to use in rose colds because it paralyzed the end organs of the vasomotor nerves. Norval Pierce used it after the removal of edematous polyps to prevent their return.

A case of chronic vasomotor rhinitis associated with perennial allergy was selected for the study of the action of this drug. Using cotton on a probe dipped into fifty per cent trichloracetic acid a streak was made on the medial surface of the turbinate extending from the posterior to the anterior end. At the end of a week the nasal stoppage had disappeared and the patient had a good clinical result, at least for the time being.

Antedating the application of the trichloracetic acid a biopsy was taken from the right middle turbinate. Thirteen days following the application of the fifty per cent trichloracetic acid a biopsy was taken from the right inferior turbinate. The first biopsy was taken from the middle turbinate and the second from the inferior turbinate in order to obviate changes in the second

biopsy due to trauma. The piece removed before the application of the acid showed the following: the epithelium was ciliated columnar and numerous goblet cells were present; the basement membrane was pale and thickened; the tunica propria was edematous and infiltrated with a large number of eosinphils together with a few plasma cells and lymphocytes; the glands were active; the blood vessel walls appeared normal.

The biopsy taken thirteen days following the application of trichloracetic acid showed the following: on the margin of the inferior turbinate there was a deep ulceration, the base of which was necrotic. The necrotic tissue and the viable tissue around it were densely infiltrated with chronic inflammatory cells. The epithelium at the edges of the ulceration was stratified squamous while that further away from the ulcer was ciliated. There was a hyperplasia of the fibrous tissue in the region of the base of the ulcer.

To study the action of the actual cautery a biopsy was taken of a patient that had been cauterized four months before because of non-allergic vasomotor rhinitis. The patient secured only partial relief from the cautery. To study the immediate action of the cautery, puncture was made of edematous tissue on the middle turbinate of a patient suffering from chronic vasomotor rhinitis and in another patient an edematous polyp was punctured with the cautery tip. The biopsy through the right inferior turbinate which had been cauterized four months before showed: the epithelium was ciliated. In it there was a depression at the base of which was an area of dense fibrous tissue. Glands in this region were distorted by scarring but were active. There was no edema in the tunica propria in the region of the scar, although at some distance from it edema was seen.

The edematous tissue on the middle turbinate seven days after the application of cautery showed: the area not cauterized was covered with ciliated columnar epithelium; the tunica propria was moderately edematous with some infiltration with plasma cells, lymphocytes and eosinophils. At the site of the cautery puncture there was a coagulation necrosis; the surrounding tunica propria was fibrous; there was a metaplasia of cells in the acini of the secretory glands; the blood vessel walls were thickened; around the site of the pucture was an infiltration of a large number of polymorphonuclear neutrophils and a few lymphocytes.

When the edematous polyp was punctured a large amount of clear fluid escaped from the puncture. Three days after the

cautery, grossly, a thick white membrane covered the polyp; the membrane was adherent to the underlying tissue. Microscopically this membrane was a thick exudate covering the area where the epithelium had been destroyed. The surface of the polyp not cauterized was lined by ciliated columnar epithelium. Beneath the exudate there was a marked infiltration of polymorphonuclear neutrophils and a few lymphocytes. The lesion produced was one of coagulation necrosis with fibroblastic proliferation in the polyp.

Cats were used for the study of the action of alcohol on the mucous membrane of the nose. The cats were anæsthetized by the intraperitoneal injection of two or three cubic centimeters of a ten per cent solution of sodium amytal. After packing off the nasopharynx with vaseline gauze, one nostril was filled with 70 per cent alcohol which was left in for twenty minutes. Dr. Robert E. Parrish (personal communication) suggested that if alcohol packs were used in the nose for allergic rhinitis they should remain in the nose twenty minutes.

In each case there was sneezing and struggling movements. In about ten minutes a bloody discharge was noted. Cats were killed at various time intervals and the nostrils sectioned and stained for histological study.

In cats killed two days after treatment the treated nostril was occluded by a red edematous mucous membrane and a serosanguinous discharge filled the treated nostril. There was hyperemia and edema to a lesser degree on the untreated side. Microscopic study showed the treated side to be partially denuded of epithelium, especially along the septum. In the recesses between the turbinates ciliated epithelial cells persisted although the nuclei stained poorly and the cytoplasms appeared granular and cloudy. Cilia were still present. Strips of epithelium were seen to be separated from the tunica propria by serous fluid (bullæ). The tunica propria contained massive areas of edema and extravasated blood. The vessels of the tunica propria were markedly engorged. The cells of the mucous glands showed cloudy swelling just as did the epithelial cells where present. No reaction was noted in the periosteum or perichondrium. Between the turbinates and septum there was a thin serous fluid containing blood pigment and scattered polymorphonuclear neutrophils.

The untreated side showed hyperemia and edema of a small degree. There was some thin acellular serous fluid between the turbinates.

Cats killed three weeks after treatment with 70 per cent alcohol showed, on the treated side, masses of granulation tissue between the turbinates and septum. This granulation tissue was deeply infiltrated with chronic inflammatory cells (polymorphonuclear neutrophils, lymphocytes and plasma cells). The epithelium covering the turbinates was, for a great part, missing, and where present was of the stratified squamous type. The tunica propria was thick; its vessels were engorged. Nothing but skeletal remains of glandular epithelium was seen. The tunica propria was infiltrated with many chronic inflammatory cells. Deep in the turbinates numerous islands of young proliferating bone could be seen.

The untreated side of the three weeks specimen was entirely normal except for a mild congestion of the small vessels.

At three months in the cats treated with 70 per cent alcohol the only gross difference noted in the treated and untreated sides was a thinning of the turbinates on the treated side. Microscopically, on the treated side, the epithelium was entirely of the stratified squamous type. Comparing the mucosa of the two sides, the treated side was found to be thinner and the tunica propria composed of denser fibrous tissue. The blood supply of the treated side was less; there were fewer glands. The bone and cartilage of the two sides were identical. The picture on the treated side was one of atrophy and fibrosis.

Summary: The instillation of 70 per cent alcohol in the noses of cats produced violent congestion and exudation with sloughing of the epithelium. This was followed by the formation of granulation tissue in the nose, a loss of glands, proliferation of bone and a metaplasia of the epithelium. In three months' time definite atrophy of the mucosa had taken place.

At the suggestion of Dr. E. C. Bryce (personal communication), edematous nasal polyps were injected with 95 per cent alcohol using a tuberculin syringe and a hypodermic needle or the needle used in the clinic for making intramucosal tests.

Clinically a decrease in size of the polyps is seen as early as the day following the injection. In one instance, two days following injection of a medium sized polyp the patient returned with no polyp tissue visible to be removed for study.

The day after injection a bright red spot is seen at the site of the injection. An ulcer may form at this point. Part of the polyp sloughs off into to nose and the remaining stump shrinks

up to form a small bud of tissue or disappears from sight in the meatus.

The usual pathological picture of a polyp six or seven days after alcohol injection is demonstrated by this case. At the site of injection is an area of complete necrosis, the necrotic area being densely infiltrated with polymorphonuclear neutrophils. The tissue superficial to this area has sloughed off leaving an ulcerated surface. Around the necrotic area is an area of hemorrhage into the tissues resembling the picture seen in an infarct. Outside this area the tissue is seen to be involved by coagulation necrosis which diminishes in severity as one leaves the site of injection. Finally, an area of cloudy swelling is seen which may or may not include the remainder of the polyp, depending on its size and the amount of alcohol injected.

To study the action of concentrated phenol on the mucous membrane of the nose, dogs were used. Dogs were anæsthetized with intraperitoneal injections of sodium amytal, using the method of Palmer (Ann. of Otol., Rhin. and Laryng., 44:25, 1935), their noses swabbed, on one side only, with a concentrated solution of carbolic acid. They were killed by air embolism at varying time intervals.

Those killed in two days showed grossly, on the treated side, a small amount of red exudate limited to the parts of the nasal cavity reached by the cotton swab. Microscopically the exudate was sero-sanguinous. In the region of the exudate the epithelium was denuded, the tunica propria was edematous and contained a small amount of extravasated blood and the vessels were deeply engorged. Small areas of hemorrhage had separated the tunica propria from the periosteum. On the whole the reaction was less violent than that seen in cats two days after instillation of alcohol into the nose. In areas not accessible to the phenol swab and on the untreated side no change whatsover was apparent.

Three weeks after the phenol swab was used much of the epithelium was still absent from the treated side. Where present it was ciliated. The mucous glands were merely dilated spaces in the tunica propria with no epithelial lining. The vessels were still dilated but did not contain much blood. The bone showed no change. The untreated side appeared normal as it did at the two day period. Early atrophy with a retarded formation of the epithelium of the glands and mucous membrane seemed evident.

In the three month specimens the treated side was lined by stratified squamous epithelium. The tunica propria was fibrous

but more vascular than the untreated side. The stratified epithelium was hyperplastic. Glands were present and appeared healthy. This is inconsistent with the picture of early atrophy seen at three weeks.

Summary: Swabbing the nasal mucosa of dogs with strong phenol solution resulted in a partial loss of epithelium, a hemorrhagic exudate and engorgement of the mucous membrane limited to the areas in contact with the solution. In three weeks what seems to be early atrophy is present, but at three months there is a definite picture of fibrosis and hyperplasia. Palmer (Ann. of Otol., Rhin. and Laryng., 44:25, 1935) reported the essential change following the use of phenol was fibrosis of the nasal membrane.

Conclusion: These observations bring us to the conclusion that if a successful result can be secured in chronic vasomotor rhinitis by treatment on an allergic, dietary or endocrine basis; or by the correction of septal deformities; or by the removal of adenoids, or the eradication of infection, etc., the desired result will be secured without deleterious changes in the mucous membrane of the nose. When we resort to ionization, the use of phenol, trichloracetic acid, alcohol, cautery, or any of the methods mentioned in the latter part of this paper, then we produce deleterious changes in the nasal mucosa.

DISCUSSION

Dr. French K. Hansel, St. Louis: Dr. Dean is to be congratulated on the excellent manner in which he has presented this material. It illustrates the necessity of laboratory and experimental procedures to determine the fundamental principles of ionization of the nasal mucosa and to estimate the value of the clinical application of this method of treatment.

In the discussions which have appeared in the literature on the nasal manifestations of allergy, a number of terms have been used to designate this condition, such as allergic, vasomotor and hyperesthetic rhinitis. Vasomotor rhinitis has been designated as of allergic and as of non-allergic origin. This is confusing, as it introduces a variety of vasomotor nasal conditions into the allergic picture. Perhaps it may be well to suggest, therefore, that all allergic conditions in the nose be indicated as nasal allergy, with further qualifications as to whether the condition is perennial or seasonal (hay fever), etc., or belong to a certain clinical or etiologic type.

In our clinical studies we have found by an analysis of all cases of suspected nasal allergy, including thereby the borderline or questionable types, that approximately 20 per cent, in children as well as in adults, do not prove to have the nasal manifestations of allergy. This non-allergic group includes a great variety of conditions, such as frequent colds and all those conditions which, as the result of disturbances in the vascular and secretory mechanisms, cause nasal obstruction and increased discharge. Accurate diagnosis, therefore, is important, for therapeutic results will not follow allergic management in these cases. As Dr. Dean has pointed out, the non-allergic vasomotor conditions of the nose respond more satisfactorily to ionization. Diagnosis must be based primarily on the clinical history and laboratory procedures, with especial reference to the cytology of the nasal secretions, skin and mucosal tests.

The treatment of allergy of the nose and paranasal sinuses must be based primarily upon allergic methods. An etiologic classification of cases must be established. Therapeutic management concerns, therefore, the following types: hay fever or pollinosis, inhalant (other than pollen), food, bacterial and combined types of any of the above. Results are obtained by avoidance of pollen, inhalants, and foods, or by desensitization to these allergens, including bacteria. Secondary matters of treatment concern the endocrines, vitamins, etc.

Local treatment of the nose and paranasal sinuses concerns the correction of anatomic deformities, the removal of nasal polyps or sinus surgery to eliminate infection and pathologic tissue. Non-specific measures, as pointed out by Dr. Dean, such as ionization and the use of escharotics may be employed in selected cases. In any event the choice of treatment must depend upon accurate diagnosis.

On the whole, Dr. Dean's discussion has given us an idea of what can be done in the laboratory to learn more about the fundamentals of diagnosis and treatment of nasal allergy, and it emphasizes the important part that allergy plays in the field of otolaryngology.

Dr. Ralph A. Fenton, Portland, Ore.: Washington University, through its outstanding research in otolaryngology, has now definitely solved many of the puzzles attending the use of various caustic agencies for the relief of allergic rhinitis. Every word of this important contribution — today much abbreviated by time limitations — should be remembered by rhinologists. Too often we are prone to listen to special pleading on behalf of such methods as ionization, forgetting that commercial interests are in the background intent on the sale of expensive apparatus.

Histopathologic evidence submitted here showing the destructive action of every sort of caustic agent employed against boggy turbinates and polyps must convince the most skeptical, especially when such patients were found still reacting to their respective allergens by intramucosal test. Realizing also how metaplasia into squamous from ciliated epithelium presages ozœna, we may anticipate, as we predicated two years ago, that such difficulties may later crop up in many instances.

Since irradiation, roentgenologic or with radium, does not damage epithelium but promotes fibrosis by killing lymphoid cells which are

phagocytized en masse with immediate development of fibroblasts, it is preferable to those agents which cause violent superficial necrosis. And while these latter may, by destroying nerve endings along the blood vessels, give temporary relief, Dr. Dean is entirely correct in his insistence upon removal of obstructions to nasal breathing, cure of sinusitis, dietary and endocrine regulation, as well as avoidance of or vaccination against the offending allergen. This is at present a complex and often difficult process. No wonder that proponents of quick and easy cures find a remunerative audience. But the last word respecting nasal allergy will not be said until efficient control through the sympathetic neurovascular mechanism can be devised.

DR. L. W. DEAN, closing: I am sorry that time would not permit Dr. Hansel to speak of the method of decentralization that we use. He is the "master mind" in this thing and the results that he gets with his hay fever patients, even if they come in at the height of the hay fever attack, are astonishing.

I have to apologize because this paper represents two years of work and it was very difficult to pick out those things that seemed to be important. I trust that those of you who are interested will make a study of it when it is published.

I think that the answer to the treatment of hay fever is first to have the patient under the care of the rhinologist as well as the allergist and make the nose normal, to study the case thoroughly from the immunological standpoint and only very, very rarely resort to some procedure which will produce fibrosis.

I am under obligation to Dr. Fenton for mentioning the influence of radium. If you will read the paper you will find in there a statement to the effect that radium leaves the epithelial cell perfectly normal while in the tunica propria you can see scattered fibroblasts which will produce fibrosis, secretion of fibrosis if that is what you wish to call a shrinking of the membrane.

The last thought that I would like to leave with you is the necessity of studying the case. I do not care whether you are an authority in allergy of national or international reputation, you are not going to get anywhere in the handling of any patient unless you study the patient and restudy him.

If we have a patient in our service with allergic rhinitis and that patient, at the end of two or three weeks, is not doing well, we do not assume that everything has been accomplished that can be accomplished; we assume that we have slipped and we go back and study and restudy the case. Frequently, we resort to the leucopenic index in these very difficult cases. It is surprising how much information you get regarding food allergy by making a study of the leucopenic index when the patient is fed this or that food.

EXTRADURAL INFLAMMATION: A STUDY OF ITS OCCURRENCE IN ACUTE SURGICAL MASTOIDITIS*

LAWRENCE R. BOIES, M.D.

MINNEAPOLIS

THIRTY-ONE years ago, Whiting, in his monograph on *The Modern Mastoid Operation*, stated that "the possibilities of surgery of the mastoid process as regards the extent of procedures upon that structure for the relief of inflammatory conditions are practically exhausted; there remains only the necessity for perfecting our technique in order that the needful steps may be accomplished with neatness and with requisite dispatch."

This confident statement apparently had been inspired by the improved end results with the technique described by the author as the "complete mastoid operation." By that time the Schwartze operation had become widely acknowledged. Additions had been made to it by Gruening who advocated a thorough removal of the mastoid tip, and by Whiting who recommended a complete exenteration of the zygoma. The addition of these steps to the basic Schwartze technique constituted this complete operation.

It seems probable from a review of Whiting's contributions that he was not not aware of the possibilities of suppuration in the petrous pyramid. Only a few years earlier (1883) Bürckner had made the first observation on suppuration of the pyramid, and Muck in 1900 by an intracapsular route, and Goris in 1903 by the subdural approach, had operated for petrous suppuration. The first anatomic studies on the cellular structure of the pyramid had been reported in 1904 by Mouret.

The importance of Whiting's emphasis on the complete operation apparently received widespread but not universal approval. Today, two characteristic attitudes exist toward the performance

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of the operation which we term simple mastoidectomy. One is an expression of the belief that all an acute surgical mastoid needs is a simple form of drainage; the other, that this condition demands a thorough complete exenteration of all cell structure.

The newer knowledge regarding petrositis which has arrived in the last five or six years has, however, again emphasized the need of a more complete mastoid surgery. As our knowledge of suppuration of the petrous pyramid has increased, it has become evident that this condition is a common concurrence to mastoid suppuration, and that a thorough complete mastoidectomy with an intracapsular exposure of cell tracts will effect a cure in most cases.

Aside, however, from the need of a thorough exposure of cell tracts in a mastoid operation to drain infection of the petrous pyramid is the consideration of an adequate drainage of the whole of an infected area in routine mastoidectomy. The great majority of complications of an acute surgical mastoiditis develop through a direct extension of infection through the mastoid structure to form an inflammatory process extradurally. From such a focus the more serious complications of bloodstream infection, meningitis, or brain abscess most often develop. It is relatively uncommon that these complications arise through infection borne by vascular channels alone without the nidus of an extradural inflammatory process.

Extradural inflammation in the routine acute surgical mastoid is more common than is usually suspected. The study which is the basis for this paper was made first to determine the incidence of this inflammation, and second to evaluate the particular anatomic factors, if any, which influenced its development at any particular site.

MATERIAL

Three hundred consecutive cases of acute surgical mastoiditis observed at the University Hospital and in the writer's private practice comprise the material of this study. These were seen in a five-year period prior to March, 1936. The spread over this period provides a material in which the virulence of any particular epidemic is mitigated as an important factor of unusual incidence. The age incidence was as follows:

TABLE I

AGE	NUMBER	PER CENT
Under 3 years	37	12.3
3 to 5 years	41	13.6
6 to 10 years	73	35.6
11 to 20 years	83	27.6
21 to 30 years	28	9.3
31 to 40 years	14	4.6
41 to 50 years	16	5.3
51 to 60 years	7	2.3
Over 60 years	1	.3

There was nothing unusual in the duration of the otitis media prior to the operation. There were a few cases in which the otitis had apparently existed less than one week; in a few it had existed over eight weeks. The longest was approximately three months. The majority were between three and six weeks.

NATURE OF OBSERVATIONS

No attempt will be made in this discussion to evaluate the two factors always present in an infection. I refer to the virulence of the infecting organism and the ability of the host to limit the infection. In the measure of their importance, judgment must be largely speculative.

Cultures of the mastoid pus were made routinely; most of the cases of mastoiditis in which the infection was manifestly severe were due to hemolytic streptococci or to pneumococci, with a predominance of the former.

The observation of infection reaching the dura in this study is based on macroscopic evidence. The manifestations were the presence of granulations or actual abscess formation, injection of the dura, or trophic changes. The trophic changes were evidenced by pallor, usually a greyish-white discoloration often with a rather wet appearance of the area involved. The bony covering of the site was usually found to have undergone a trophic change. These characteristics are probably evidence of an infection of only a moderate severity.

These changes were noted at two sites: at the lateral sinus and over the middle fossa above the tegmen of the mastoid, the antrum and the tympanic cavity. In seven cases of petrositis it

was assumed that the inflammation had actually reached the dura because of the presence of abducens paralysis in six of the cases, and a severe retro-ocular pain in the other.

Observations on the anatomic factor were likewise macroscopic. Two conditions seem to influence the occurrence of extradural inflammation. One is the matter of pathological pneumatization with the persistence of tissue or the arrangement of structure making the spread of infection to the dura more likely; and the other, the extension of pneumatic cells to sites where drainage from infection if present is inefficient, particularly when that cell structure comes into a close relationship to the dura bordering on the mastoid surfaces.

Gross evidence of pathological pneumatization as observed in the course of performance of operation for acute surgical mastoiditis can be determined by two unmistakable signs. One is the small under-developed mastoid without any appreciable cell structure, and the other the matter of sclerosis with only a partial development. Sclerosis, of course, may appear in a structure which had been normally pneumatized.

The spread of infection in sclerotic types of mastoids is probably influenced by channels which lead to the inner table, and the fact that a collection of pus could easily be under pressure; the latter influenced by a thick cortex and the distribution of sclerotic bone. The persistence of vascular communications in arrested pneumatization which predispose to the spread of infection at certain sites would not be detected by gross observation. Braun describes the principal sites as:

(1) At the junction of the tegmen antri and tegmen tympani, in front of the convexity of the superior semicircular canal;

(2) On the posterior surface of the petrous pyramid, above and lateral to the mouth of the aqueductus vestibuli; and

(3) On the floor of the middle ear.

Abnormal arrangement of structure, making the spread of infection to the dura more likely, may be present in the form of a forward position of the lateral sinus, or in the position of the tegmen of the antrum. The distance of the knee of the sinus from the posterior canal wall has been measured from one to twenty-five millimeters (Braun). It would be reasonable to consider the sinus as abnormally forward if the distance in the mastoid at an age when pneumatization is complete is twelve millimeters or less. An abnormally low-lying tegmen is present when

there is no development of cells below it and the space of the antrum.

In the matter of the extension of pneumatic cells, three locations are important aside from the development in the petrous pyramid. These are an extensive development in the squama or zygoma, a well-developed extension of cells in the postero-superior angle, and a large tip with cells deep to the digastric crest.

The size of the cell is undoubtedly important. In Bezold and Siebenmann's *Textbook of Otology*, this statement is made: "The importance of the size of each single cell for the absorption of pus becomes evident if you remember that the surface of a ball increases in proportion to the square of its diameter, while its contents to the cube. The power of absorption of the walls decreases therefore with the increase of the size of the cell. The smaller the single cells are the easier they will master the pus they contain and the number of cells that are inflamed is immaterial."

INCIDENCE OF EXTRADURAL INFLAMMATION

In the three hundred cases there was macroscopic evidence of the inflammation actually having reached the dura in ninety-eight instances. Seven cases of petrositis in which clinical symptoms or signs indicated an extradural inflammation was present bring the total to one hundred and five. This produces an incidence of thirty-five per cent.

The distribution as to site was as follows:

	NUMBER	PER CENT
The lateral sinus	64	21.6
The middle fossa	17	5.6
Both lateral sinus and middle fossa	17	5.6
The surface of the petrous pyramid	7	2.3

In the instances where the middle fossa dura and the lateral sinus were both involved, the two areas were adjacent, indicating that one was an extension of the other.

CHARACTER OF THE LOCAL CHANGE PRODUCED BY THE INFLAMMATION

The character of the evidence of this inflammation has been discussed. The incidence was noted as follows:

	Granulations with or without actual abscess formation	Injection or trophic changes
	NUMBER	NUMBER
Involvement of sinus	40	24
Involvement of middle fossa	5	12

Involvement of both sinus and middle fossa dura occurred in seventeen cases as follows:

Granulations on sinus, injection or trophic changes on dura	6
Granulations on both	2
Granulations on dura, injection or trophic changes over sinus	4
Injection or trophic changes on sinus and dura	5

ANATOMIC FACTOR IN THE DEVELOPMENT OF EXTRADURAL INFLAMMATION

In this evaluation of the anatomic factor influencing the occurrence of extradural inflammation it is necessary to consider the time element. The majority of operations in acute surgical mastoiditis do not occur in less than three weeks after the onset of an acute otitis. When operation is done in less than three weeks, it is usually because there has been a more or less complete destruction with post-aural edema or abscess, or the onset of signs of sepsis, or significant pain develops, indicative of dural irritation.

When the involvement of the mastoid structure is diffuse, it is obvious that any particular gross anatomic factor would seem an uncertain influence in the occurrence of extradural inflammation at any particular site. Observation of a particular portion of the mastoid as a site of more advanced destruction in cases of relatively short duration might reasonably be considered as due to a gross anatomic influence if some unusual anatomic factor seemed to be present. In the three hundred cases studied, therefore, those are eliminated from consideration in which the otitis had existed over three weeks, and those cases with an otitis of less than three weeks in which no apparently significant factor seemed obvious. This limits us to a relatively small group.

In the sixty-four cases of mastoiditis in which the inflammation actually reached the dural covering of the lateral sinus, the otitis had been present over three weeks in twenty-six of the cases; and in eleven cases in which mastoidectomy had been done in less than three weeks from the onset of symptoms, there was no apparent gross anatomic factor. This leaves twenty-seven cases. In these the development of extradural inflammation seemed influenced as follows:

By an abnormally forward position of the knee of the sinus	16
By extensive tip development with large cells in close relationship to the lower portion of the sinus	4
By extensive postero-superior angle cells	7

(Total 27 cases; 42.1 per cent of the 64 cases; 9 per cent of the 300 cases)

In the seventeen cases in which extradural inflammation was observed over the dura of the middle fossa, there were five cases in which the otitis had been present over three weeks. In the other twelve cases, the site of occurrence seemed influenced by the following anatomic factor:

Extensive cell development in the zygoma or squama or both..... 9
Poor development with a low tegmen..... 3

(Total 12 cases; 70 per cent of the 17 cases; 4 per cent of the 300 cases)

In the seventeen cases in which the extradural inflammation was observed in two sites, over the dural covering of the sinus and of the middle fossa, in thirteen of the cases the otitis had existed over three weeks, in three cases an anatomic factor could not be accounted for, and in one case the site seemed influenced by the presence of deeply developed postero-superior angle cells.

A composite chart of these items is presented in Table II.

TABLE II

	LATERAL SINUS	MIDDLE FOSSA DURA	LATERAL SINUS AND MIDDLE FOSSA DURA	SURFACE OF PETROUS PYRAMID	TOTAL
Number	64	17	17	7	105
Per cent	21.6	5.6	5.6	2.3	35.3
Character of change:					
Granulations with or without actual abscess formation	40	5	5		50
Injection or trophic changes	24	12	12		48
Duration of otitis:					
Over three weeks	26	5	13		44
Less than three weeks	38	12	4		54
Probable anatomic factor in cases with less than three weeks of otitis:					
Forward sinus wall	16				
Extensive tip development	4				
Angle cells	7		1		
Low tegmen		3			
Cell development in zygoma or squama or both		9			
No apparent anatomic factor	11		3		

The incidence of extradural inflammation according to age group was as follows:

TABLE III

AGE	NUMBER OF CASES	PER CENT	INCIDENCE OF EXTRADURAL INFLAMMATION	PER CENT
Under 3 years ...	37	12.3	4	3.8
3 to 5 years	41	13.7	12	11.3
6 to 10 years	73	24.3	27	25.7
11 to 20 years	83	27.7	39	37.1
21 to 30 years	28	9.3	9	8.6
31 to 40 years	14	4.7	6	5.7
41 to 50 years	16	5.3	4	3.8
51 to 60 years	7	2.3	4	3.8
Over 60 years	1	.3	0	0

These figures suggest an agreement with Meltzer's statistics on thrombosis of the lateral sinus in which he found that "the preponderance of cases of thrombosis occurred in patients between the ages of 6 and 30 years, the majority of cases appearing in those between 11 and 20 years. The infrequency of thrombosis of the lateral sinus in the young may be explained on the basis of the position of the lateral sinus, which plays a less important role in relation to the mastoid cells."

Some additional statistics on these three hundred cases are pertinent to this study. There were twelve deaths, a mortality of four per cent. Three of the twelve fatal cases were admitted in a condition beyond reasonable hope of surgical aid; one with an extensive acute brain abscess; another with a pneumococcus type III meningitis; and the third with a lateral sinus thrombosis complicated with a streptococcus meningitis. Eight of the other nine cases died from meningitis which complicated a blood stream infection in four cases, petrositis in two cases, and a labyrinthitis in one. There was one death from sepsis from an extensive osteomyelitis of skeletal bones complicating a blood stream infection.

There were twenty-one cases presenting the clinical picture of a well-established sepsis from invasion of the blood stream by way of the lateral sinus. Four of the cases were fatal, a mortality of nineteen per cent. Jugular ligation was done in twelve of the cases with one death in these twelve. It is not within the scope of this paper to discuss the treatment of lateral sinus thrombophlebitis. These twenty-one cases were characterized as instances of well-established sepsis. In the total of sixty-four cases in which it was observed that inflammation actually reached

the dural covering of the sinus, a brief septic temperature with a single chill or two had been present in a number of instances and was cut short by a complete mastoidectomy with exposure and drainage of the site of disease. It is our belief that when an actual thrombophlebitis is evident and a complete mastoidectomy with adequate exposure of the diseased site has not brought the desired improvement, that incision of the thrombosed site and removal of the infected area is the next step. In the event the sepsis continues, jugular ligation is in order if there is evidence of infection in the bulbar end of the sinus.

This method of management was, I believe, first recommended by Lillie. Our mortality figures approach the low percentage he has reported.

There were no cases in this series presenting the clinical picture of an acute hemorrhagic mastoiditis as described by Kopetzky, namely, signs of sepsis concurrent with the onset of the mastoiditis, intact bony intercellular walls at operation without any detritus or pus, and a thickened swollen mucous membrane which bled easily.

In the matter of criteria of complete mastoidectomy, I am in agreement with the requirements expressed by Rosenwasser and Druss as the teaching of Friesner: "At the end of the complete operation, the surgeon should be able to see clearly the external semicircular canal, a clean initial groove, a clean sinus plate, the middle fossa plate, the digastric muscle, the mastoid tip removed, and a cleanly exenterated zygoma." These were requirements originally taught to me by Meltzer in the graduate courses in otology at Harvard and at the Massachusetts Eye and Ear Infirmary.

We often find in the performance of a complete mastoidectomy that the sinus or middle fossa plate has been thinned to such an extent that exposure is necessary. It is our policy to intentionally expose the dura over the sinus or the middle fossa or both when the symptoms or signs prior to operation are not adequately explained by the findings when the mastoidectomy is done. In the three hundred cases of this series, healthy exposure was made as follows:

Lateral sinus	26
Middle fossa	16
Both lateral sinus and middle fossa	22
With disease at another site	16
Total	80

This means that in a total of 178 cases (59.3 per cent) of this series dural covering was exposed. I have never observed an ill effect from this exposure. It is conceivable that postoperative retention of purulent secretion in the mastoid cavity as influenced by the packing might be a definite influence in the infection of this exposed dura.

SUMMARY

A study of three hundred consecutive cases of acute surgical mastoiditis occurring over a five-year period and with a normal age distribution indicates that inflammation actually reached the dural covering of the lateral sinus or the middle fossa or along the surface of the petrous pyramid in thirty-five per cent of cases. The involvement was at the lateral sinus in twenty-one and six-tenths per cent (21.6%), and over the middle fossa in five and six-tenths per cent (5.6%). Both lateral sinus and middle fossa were involved in an additional five and six-tenths per cent (5.6%). There was evidence that the dura along the surface of the petrous pyramid was involved in two and three-tenths per cent (2.3%) of the cases.

An anatomic factor was observed in a limited number of cases to be an apparent influence in the development of the inflammation at the particular site noted. This observation was limited to those cases with a relatively short duration of the otitis in which there was not a diffuse involvement of the mastoid structure, but rather a localized area of maximum destruction, and the presence of some unusual anatomic arrangement at this site.

The incidence of inflammation reaching the lateral sinus seemed to be influenced in these cases by the following anatomic factors, in order of frequency: (1) An abnormally forward position of the lateral sinus; (2) Extensive development of posterosuperior angle cells; and (3) Large cells at the tip coming into close relationship to the lateral sinus.

The anatomic influence to inflammation reaching the middle fossa seemed influenced chiefly by extensive pneumatization in the zygoma or squama or both, and to a less extent by a low tegmen.

The high incidence of extradural inflammation emphasizes the need of a complete mastoidectomy. In this series, this required, in over one-half the cases, an exposure of the dural covering of the lateral sinus or of the middle fossa or both.

The mortality in this series was four per cent including three instances in which the patients were admitted in a condition beyond reasonable hope of surgical aid.

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DISCUSSION

Dr. H. P. Mosher, Boston: This is a short, crisp paper which keeps very much to its subject.

The historical introduction carries a lesson, namely, not to think what you do is the last word in anything. The writer quotes Whiting as saying, after he had given his technique for mastoidectomy, "This is the complete mastoid operation." "Operating procedures on the mastoid are practically exhausted." In his opinion all that remained was to acquire facility in operating technique.

Even before Whiting wrote these confident words, observations had been made on suppuration of the petrous apex (1883 Bürckner). Fifty years ago, and even thirty years ago, operating approaches to the petrous tip had been described. Therefore, the mastoid operation of Whiting was not complete and I venture to say it is not complete today even with the advent of Kopetzky and Eagleton.

Following his historical introduction with its amusing sidelight the writer states that the great majority of complications of mastoiditis are

due to direct extension of the infection to the dura by bone. He believes that the infection is infrequently carried by vascular channels. The more I have to deal with infection in the skull the more I feel that the vascular route, especially the venous route is the more important. Histological study shows this to be especially true of brain abscess. It is true also of osteomyelitis.

We instinctively think of the dura as a protective organ. It furnishes protection both against trauma and against infection. It attempts to stop infection by the formation of extra or subdural abscess and with the help of surgery often succeeds. We long ago learned that the dura can be exposed with impunity over the lateral sinus and the middle fossa. In doubtful cases, no mastoid operation can be called complete unless such an exploration is made.

We are indebted to Hann, of Toronto, for a bit of new knowledge on the dura. It consists of three layers, instead of two, as we have always thought, and between the two inner layers is a capillary bed of vessels. This capillary bed is often ruptured by trauma. I believe it must play a part in subdural infection and its spread.

To speak again of the protecting function of the dura, we all know that there may be an infection in or about the lateral sinus and to a less extent on the dura and nothing further happens.

I am indebted to Dr. Boies for working out in his series of 300 cases the percentage of cases in which the dura was involved, namely, in 35. Twenty-one per cent were involvement of the lateral sinus, 5 per cent of the dura of the middle fossa, and 2 per cent of the dura of the petrous apex. This is the first time we have had actual figures. They are no surprise because they bear out classical findings which have long been known. Nevertheless they have a distinct practical as well as an academic value.

The writer gives obvious and satisfactory anatomical reasons for the difference in the location of involvement of the dura.

Table III giving the age incidence of lateral sinus thrombosis (6 to 30 years) and its agreement with Meltzer's statistics is comforting.

In dealing with thrombosis of the lateral sinus, the writer opens the sinus and waits for continued sepsis before tying the jugular vein. This procedure was advocated by Kopetzky some years ago and much can be said in its favor. It has succeeded and will succeed again. If, however, in any given case, metastasis promptly occurs and you have not tied the vein, the operator cannot help feeling a bit guilty. Anyway, I am old-fashioned enough to feel guilty in such a case.

The writer quotes Friesner as to the technique of the "complete mastoid operation" which calls for the routine removal of the mastoid tip. I followed this practice for some years and then gave it up except where the tip was obviously diseased. I have had no reason to change back.

To me, the paper is refreshing because, as I said in the beginning, it is short, crisp and keeps to the point. It gives statistics which are new, at least to me. The facts which are tabulated are not new. To know that a thing happens is important. To work out how often it happens is much more important.

DR. LAWRENCE R. BOIES, closing: Perhaps I left the wrong impression in my statement regarding the spread of infection by vascular channels. I feel that the blood vessels play a very important part in the spread of inflammation through the bone. However, in my experience I have found that in the majority of instances where sinus phlebitis, meningitis, or brain abscess has been encountered, if the dura is exposed over the site of involvement one will usually find that there is evidence that the bone is diseased contiguous to this dura and that there is evidence of an extradural inflammatory process.

I referred to seven cases of petrositis. There were two additional cases in which the diagnosis was made by the X-ray and operative findings, but in which pain was a minor symptom and no abducens paralysis was present. These nine cases make an incidence of three per cent for petrositis in this series.

TUMORS AND CYSTS OF THE MOUTH AND JAWS OF INTEREST TO THE OTOLARYNGOLOGIST

ROBERT H. IVY, M.D., D.D.S., F.A.C.S.

PHILADELPHIA

THE MOUTH and the upper respiratory passages are so closely related anatomically that practitioners in these respective fields should have a fairly intimate acquaintanceship with the pathological conditions of both, at least from the standpoint of diagnosis. Nose and throat specialists are constantly being consulted about abnormalities involving the oral cavity and jaw bones, so that a discussion of some of these is therefore not out of place. We shall limit ourselves to new growths and allied conditions. In the upper jaw, especially, there are certain tumors and cysts which, on account of possible complications in the nose and maxillary sinus, may be of special concern to the otolaryngologist. We shall not dwell on malignant tumors — carcinoma and sarcoma — important as they are, as too much time would be required. The largest group with which we shall deal is that consisting of the tumors and cysts depending for their origin on abnormal growth of cells concerned in tooth development.

Three types of dento-cystic tumors are described — the adamantinoma, the dentigerous cyst and the dental root cyst. Reference to the appended chart (Fig. 1) will give the essential features of each of these.

(1) The *adamantinoma* or *ameloblastoma* is the least common as well as the most serious of the three forms of dento-cystic tumor. It is believed to be due to aberrant growth of the ameloblasts or enamel-forming cells of the tooth germ, although there is never any calcified enamel in the tumor. The lower jaw is usually involved, but the upper jaw may be affected. The most common site is the molar region of the mandible. The tumor causes a distention of the bone, a cavity being formed containing

FIG. 1 — Epithelial Tumors and Cysts of the Dental System

NAME	ORIGIN	PATHOLOGIC ANATOMY	X-RAY	AGE	CLINICAL CHARACTERISTICS	PROGNOSIS	TREATMENT
1. Ameloblastoma (Adamantinoma)	Aberrant growth of epithelial cells which are prototypes of those forming inner layer of enamel organ (ameloblasts).	Cavity in bone divided into numerous compartments by fibrous or bony septa. Some of the spaces are cystic, containing fluid, others are filled with solid tissue. Epithelium is cuboid or columnar, and arranged in strands or alveoli, surrounding a stellate reticulum.	Cavity in bone divided by fine trabeculæ into compartments varying in size.	Any age.	Painless expansion of bone, usually in molar region of mandible, slowly increasing in size. May be an opening into mouth from cystic cavity.	Recurrence common unless radical operation is done.	For small growths shelling out and curettement may suffice. For larger growths, complete resection of portion of mandible involved.
2. Dentigerous (follicular) cyst	Cyst lining formed by epithelium of outer layer of enamel organ.	Single cavity in bone. Membrane is lined with layers of squamous epithelium. Contains thin fluid and the crown of an unerupted tooth projects into the cyst from its bony wall.	Clear area of absence of bone, with well-defined margins, containing an unerupted tooth	Any age, but particularly about time of eruption of permanent teeth.	Slowly increasing expansion of a part of the bone, which later may become very thin and give parchment or celluloid feeling. Absence of a tooth from the series in the region of the swelling is significant.	Readily cured by suitable operation.	Operation through flap of gum. Complete removal of cyst with its capsule, together with unerupted tooth.
3. Dentoperiosteal (dental root) cyst	Abnormal growth of epithelial cell-rests normally present in dental periosteum, derived originally from outer epithelial layer of enamel organ (paradental epithelial rests of Malassez).	Cavity in bone about apex of permanent tooth whose pulp has been devitalized. Cavity lined with multiple layers of squamous epithelium and contains fluid with cholesterin crystals.	Radiolucent area in bone with clearly defined margins in connection with apex of pulpless tooth or in part of bone from which pulpless tooth has been removed.	Adults.	Presence of pulpless tooth or history of previous removal of such a tooth. Slowly increasing painless expansion of bone. Puncture allows discharge of clear fluid or pus if secondary infection is present.	Readily cured by suitable operation.	Flap made in gum. Removal of offending tooth if present. Epithelial lining shelled out in most cases.

a lobular mass, or there may be several cavities divided by fibrous or bony septa. In the earlier stages the growth may consist largely of solid tissue, but numerous cysts are formed later, finally coalescing into one or two large cysts filled with a viscid yellowish or brownish fluid. Histologically, there are nests of columnar and cuboidal epithelium, corresponding to ameloblasts, surrounding less dense areas with compressed cell bodies resembling the stellate reticulum of the enamel organ. These epithelial areas are surrounded by a supporting connective tissue containing blood vessels. The surrounding bone may be gradually absorbed, the tumor cells then breaking through and invading the surrounding soft tissues. Metastases, however, are practically unknown. In general, it may be said that the tumor is to be regarded as benign, but requiring radical treatment on account of its local infiltrative characteristics. It may be found at any age.

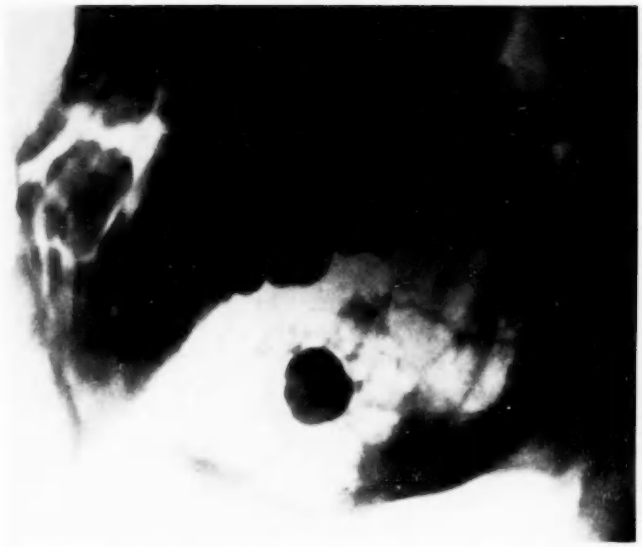


FIG. 2 — (Ivy) — Radiograph of adamantinoma involving practically half of mandible.

SYMPTOMS AND DIAGNOSIS. The patient presents a painless swelling, usually in the molar region of the mandible, which has been slowly increasing in size. The jaw is found to be expanded, the outer plate being more affected than the inner. When the tumor has broken through the bone, it may give a lobulated surface to the overlying mucous membrane or skin, and an elastic

feeling on pressure. If an opening into a cystic cavity exists, a fluid discharge is seen that is usually viscid and brownish. Infection may occur and obscure the nature of the tumor by symptoms of osteomyelitis. The diagnosis from purely inflammatory conditions and from other tumors and cysts can usually be completed by the X-ray picture, which shows a cavity in the bone, divided by fine bony trabeculæ into numerous compartments (Fig. 2). Sometimes, in the more solid form, histologic examination is necessary in order to differentiate the growth.

FIG. 3 — (Ivy) — Appearance of adamantinoma of upper jaw.

TREATMENT. In early cases in which the tumor is small and surrounded by well-defined bony walls, it is sometimes possible to obtain a cure by enucleation and curettement from within the mouth, followed by implantation of radium. If the tumor is large, with irregular extensions into the surrounding bone or perforation into the soft tissues, complete resection should be the initial treatment. Of sixteen cases treated by the writer, four are believed to be well after conservative enucleation, repeated several times in one case. Two patients are apparently well after a second conservative operation followed by implantation of radium. In four cases, complete resection with loss of continuity of the mandible was the initial treatment, with complete cure. Six patients after conservative operations have finally come to radical resection, with apparent cure. In one case, the upper jaw was involved, and responded to enucleation (Figs. 3, 4 and 5).

FIG. 4 — (Ivy) — Radiograph of case of adamantinoma shown in Fig. 3. Radiographically this resembles a dentigerous cyst owing to presence of unerupted canine tooth, but bone cavity contained solid tissue instead of cystic fluid.

FIG. 5 — (Ivy) — Radiograph of case shown in Figs. 3 and 4, one year after enucleation of tumor tissue, showing regeneration of bone to fill cavity formerly occupied by tumor.

(2) The *dentigerous* cyst takes its name from the fact that it *bears a tooth* and forms a hollow swelling usually filled with straw-colored fluid and having a partially or fully developed tooth attached by its root portion to the wall of the cyst, with the crown projecting into the cavity. There is a lining membrane, the inner layer of which is of epithelium derived from cells of the enamel organ. These cysts are usually first noticed about the period of eruption, nearly always in connection with the permanent teeth. Any tooth may be involved, most commonly the canine, and then the third molar, or a supernumerary tooth may be connected with the cyst. From the foregoing, it is evident that the term dentigerous should not be applied indiscriminately to all cysts of dental origin, as is frequently noticed in the literature, but only to those having the crown of a tooth in the cavity. In the upper jaw, a dentigerous cyst may expand and occupy the space normally belonging to the maxillary sinus, pushing up the floor of the latter, but rarely breaking into it spontaneously (Fig. 6).

FIG. 6 — (Ivy) — Dentigerous cyst of maxilla occupying part of space of maxillary sinus, showing unerupted third molar tooth.

SYMPTOMS AND DIAGNOSIS. There is a gradually increasing swelling of the jaw bone, usually involving the outer plate especially. Later, the bone becomes very thin, giving the characteristic parchment or celluloid feeling. The absence of a tooth from the series, with no history of extraction, is a point of great signifi-

cance. Suppuration may occur. The radiograph shows a clear area indicating a space in the bone, with well-defined margins, containing the unerupted tooth. In the upper jaw, the X-ray may show the thin outline of the wall of the cyst forming a dome-like projection into the maxillary sinus.

TREATMENT. This consists in complete removal of the cyst capsule through an incision in the gum, together with extraction of the unerupted tooth, and allowing the bone cavity to fill in by granulation. Small cysts, especially in the upper jaw, may sometimes be treated after enucleation of the lining by suturing the gum flap and allowing the cavity to fill in by organization of aseptic blood clot. The rhinologist occasionally treats such a cyst which has involved a large portion of the space of the maxillary sinus through the nose, removing the intervening partition between the cyst and the maxillary sinus, leaving the cyst lining, thus converting the whole into a maxillary sinus. We prefer to attack all cases through the vestibule of the mouth, in this way permitting a more direct view of the pathological condition present, easy removal of the unerupted tooth, and preserving the maxillary sinus intact if possible.

(3) *The dental root cyst*, or dentoperiosteal cyst, is by far the most common form of cyst found about the jaws. Its occurrence is almost always a sequel to chronic inflammation about the apex of a tooth as a result of infection following death of the dental pulp. Cysts of this type, therefore, are most commonly found in adults, and in connection with the roots of pulpless teeth. The responsible tooth may have been removed several years previously, yet the cyst may continue to grow in the edentulous portion of the jaw. The epithelial lining of the cysts is believed by most writers to be derived from the paradental epitheliel cell-rests of Malassez, which are remnants of the cells of the enamel organ persisting normally in the dental periosteum. The wall of the fully developed cyst consists of a dense fibrous capsule lined generally with several layers of squamous or cuboidal epithelium. The material in the cyst cavity may be like soft butter in consistency, or it may be a clear, straw-colored fluid, with cholesterin crystals. Secondary infection may convert the contents into pus. Dental root cysts vary considerably in size, from that of a small pea to a hen's egg. In the maxilla, like the dentigerous cysts, they may encroach on the maxillary sinus or the nasal fossa,

but they rarely rupture into these cavities (Fig. 7). We have seen many times a cyst of this type push up the floor of the maxillary sinus until the latter has been entirely obliterated, yet without any communication existing between the cyst cavity and the sinus. Most of the cysts described as arising in the wall of the maxillary sinus in reality belong to this group. By extension, a cyst may gradually involve the roots of adjacent teeth, causing secondary pulp devitalization.

FIG. 7 — (Ivy) — Dental root cyst connected with maxillary second incisor tooth, pushing up floor of nose.

SYMPTOMS AND DIAGNOSIS. Small dental root cysts may cause no symptoms whatever, the diagnosis being made by roentgen ray examination of the apical region of a pulpless tooth. This will reveal a radiolucent area with clearly defined condensed margins. Larger cysts cause a bulging of the bone about the apex of a pulpless tooth, usually labially or buccally, or in a location from which a tooth has been lost. The maxillary second incisor is the most frequent seat of involvement. If rupture has occurred, there will be a discharge of clear fluid or pus from time to time into the mouth. There is usually no pain, unless acute secondary infection occurs. Cysts which have not discharged fluid must be carefully differentiated from sarcomatous or other solid tumors. Close examination of the roentgenogram will usually make the distinction. In cases of doubt, needle puncture under aseptic

conditions will establish the diagnosis. In edentulous regions of the upper jaw, it may be difficult to differentiate in the X-ray film the wall of a cyst from the floor of the maxillary sinus. Here again, needle puncture through the gum is a valuable aid in diagnosis. The roentgenographic shadow of the cyst may overlap the shadow of the roots of nearby teeth. Here, it is important to test these teeth for pulp vitality. If they are devitalized, the success of treatment of the cyst may be jeopardized by persisting infection from these teeth. On the other hand, if pulps are found to be vital, needless sacrifice of useful teeth will be avoided.

Treatment. The practice of simply making an incision into the swelling to provide drainage of the dental root cyst, unless acute suppuration is present, is to be condemned. This never gives permanent relief, but usually introduces infection into a perviously sterile region and by the formation of adhesions of the overlying mucous membrane, renders proper operation more difficult. The responsible tooth, if still present, must usually be dealt with by extraction, and any secondarily devitalized adjoining teeth whose roots are exposed in the cyst cavity should be removed at the same time. The cyst lining is then usually shelled out completely, and the bone cavity dealt with as in the case of a dentigerous cyst. In a large cyst of the upper jaw, with a very thin partition separating it from the maxillary sinus, complete shelling out of the lining might make an undesirable communication with the maxillary sinus. Here, it is often advisable to make a very wide opening from the vestibule of the mouth into the cyst cavity, leaving the lining intact. The opening should be made large enough to remain permanent, so that the epithelial lined cyst cavity will gradually shrink from behind and eventually remain as a depression in the vestibule of the mouth. In all of these operations, local anesthesia by nerve blocking can usually be employed, with the great advantage of practically eliminating operative hemorrhage.

CALCIFIED DENTAL ANOMALIES

In addition to the cystic and soft-tissue growths originating in the dental epithelium, calcified masses of dental tissue, usually called odontomas, are not infrequent. In the commonest form, the calcified tissues are irregularly intermingled and have no def-

inite shape. Here we have a disordered conglomeration of enamel, dentine and cementum. The mass may be erupted or unerupted. These growths may be discovered at any age, and are frequently not recognized until supervening infection causes symptoms of osteomyelitis, when the hard masses are often mistaken for necrotic bone. They are found in either jaw, perhaps more often in the lower, and generally in the molar region. In the upper jaw, such a mass may encroach on the space of the maxillary sinus. Frequently there is a normal unerupted tooth beneath the anomalous mass. An irregular swelling of the bone that has existed for a long period and has practically not changed in size, in the absence of a tooth, will suggest a calcified dental anomaly. The radiograph will always reveal the nature of the trouble (Fig. 8). If a fistula is present and a probe is passed, the hard, dense character of the tissue is felt to be quite distinct from bone.

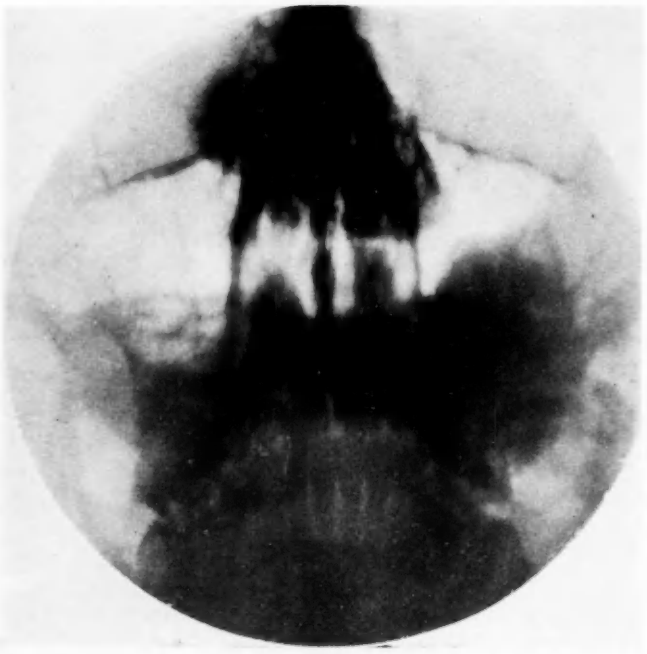


FIG. 8 — (Ivy) — Calcified dental anomaly, partially occupying space of maxillary sinus, with unerupted second molar tooth above it.

TREATMENT. If acute symptoms of infection are present, with swelling, redness and edema of the tissues of the face, and suppuration from the bone, early treatment should consist in incision and drainage. After acute symptoms have subsided, the growth should be removed, usually from within the mouth. It may be necessary to turn back the overlying gum and cut away some of the bony covering, just as in the case of an impacted tooth, in order to dislodge the mass.

NASOPALATINE CYSTS

Cysts developing in the interincisive region of the maxilla have been described by Meyer, Schroff, Thoma, Goodman and others, and should be of especial interest to the rhinologist owing to their intimate relationship to the floor of the nose. They probably arise from epithelial remnants of the nasopalatine duct. When large, such a cyst may present a swelling in the hard palate in the region of the interincisive papilla, but may only be detected

FIG. 9 — (Ivy) — Large cyst of naso-palatine canal.

by X-ray examination, which shows a well-defined radiolucent area above and between the apices of the maxillary first incisors (Fig. 9). These areas, when small, have commonly been regarded as simple enlargements of the incisive fossa. Sometimes secondary infection occurs, with discharge of pus through an opening in the palatal mucosa, usually at the site of the interincisive papilla.

A cyst of this type may be differentiated from a dental root cyst connected with a first incisor by the fact that the pulps of these teeth may be vital, and on making X-ray films in different positions the relationship of the cyst shadow to the apices of the teeth will vary.

TREATMENT. The cyst is usually best approached by laying back a flap of gum on the palatal aspect of the first incisors. Sufficient bone is removed with chisel and rongeur forceps to expose the cyst cavity, and if possible the epithelial lining is then shelled out. This lining sometimes extends in a narrow channel high up toward the nose, surrounding the nerve and blood vessels in the canal, consequently it may be very difficult to remove completely. To insure its destruction, the operation may be followed by application of Cutler-Zollinger sclerosing solution.

FIG. 10 — (Ivy) — Dentoperiosteal fibroma (epulis).

EPULIS

Objections have been raised to the use of this term because it implies any growth on the gum. It has come to be applied, however, to a definite clinical entity, the characteristics of which are: circumscribed growth from the alveolar ridge at the gingival margin of the teeth, having generally a rather narrow pedicle

and spreading thence over the gum surface, often displacing or covering the teeth (Fig. 10). These growths all spring from the dental periosteum. They may start in chronic inflammation, from irritation of food, or tartar deposits at the gum margin, ill-fitting artificial crowns and fillings, or carious cavities. Some pathologists claim that, histologically, at least, they are not tumors at all, but should be classified as infectious granulomata, and others regard them as expressions of various stages of osteitis fibrosa. However this may be, clinically and grossly they have most of the characteristics of tumors, and for the present we prefer to include them in the tumor group.

These dentoperiosteal tumors or epulides may be classified into three principal varieties, both clinically and pathologically: (1) fibroma, (2) fibroangioma, (3) benign giant cell tumors.

Fibroma. The fibroma is the most prevalent form. Soft at first, it appears later as a hard nodular mass upon the surface of the gum or pushing out between two teeth, sometimes displacing the teeth, with a clear line of demarcation where the bulging of the new growth extends beyond the normal contour of the gum. In color, the mucous membrane covering the tumor may be slightly reddened, though it often differs but little from that of the normal gum. Fibromas are almost always slow in growth, altering in size but little in months or even years. Microscopically, we see an outer layer of normal stratified squamous epithelium and submucosa, while the deeper portion of the growth consists of an interlacing network of fibrous tissue with round-cell infiltration. Sometimes there is evidence of bone formation.

Fibroangioma. This form of epulis is usually softer, having more of a spongy character than the pure fibroma. It grows more rapidly, the overlying gum is bright red in color, and it bleeds easily. Microscopically, under the covering squamous epithelium is seen a layer of fibrous tissue containing numerous capillary blood vessels.

Benign giant cell tumor. The clinical characteristics of a growth of this type are a smooth, soft, dark red or purplish swelling on the gum surface, with a broad or narrow pedicle, loosening or displacing the teeth, and increasing rather rapidly in size. Microscopically, beneath the covering mucous membrane there is a stroma of fibrocellular tissue and scattered throughout are giant cells, which vary in size and have numerous nuclei grouped toward the center of the cell. Small masses of bone

and cartilage may be found. The term commonly used formerly for these growths was giant cell sarcoma, but the word sarcoma implies malignancy, and these growths are not malignant. The giant cell present in the epulis differs from that in the true giant cell sarcoma of the malignant type. The unfortunate application of the term sarcoma to the ordinary giant cell tumors of the jaw has led to much unnecessary sacrifice of tissue, deformity and functional disability by the performance of unjustified radical operations.

Treatment of Epulis. The great majority of cases of all types just described spring from the alveolodental periosteum about the neck of a tooth, and any form of treatment that does not include removal of the point of origin is likely to be followed by recurrence. Many patients come with the history that a dentist has cut off the growth or burned it away with caustic a few weeks or months before, but that the tumor rapidly reappeared. Occasionally, attempts to destroy the growth with radium have been seen, with no consideration being given to involvement of the teeth. This results in an entirely unnecessary, slow-healing, painful ulceration, perhaps with exposure and necrosis of a large area of bone and loosening of adjacent teeth. The best method of treatment is by excision, together with removal of the tooth or teeth from the periosteum of which the growth arises. Under local anesthesia, an incision in the healthy gum is made, just outside the limits of the growth, down to the bone. Through this incision the tumor is freed as much as possible from its soft tissue attachments. The tooth most likely to be involved is then extracted, and the tumor may come away, attached to the neck of the tooth by the dental periosteum. If not, the tooth on the other side of the tumor should be removed. The bony margins of the tooth sockets are trimmed smooth with rongeur forceps. In some cases it is possible to fashion a flap of healthy gum and suture it over the exposed bone surface, thus insuring rapid healing.

The *benign giant cell tumor* may occupy the interior of the bone, the lower jaw being more frequently involved than the upper, with an expansion of the inner and outer plates. If the cortical plate becomes perforated by the tumor, the mass will have the typical purplish color seen in the giant cell epulis. Otherwise, absolute diagnosis may not be possible before operation. On incision, these growths have a dark red color, as contrasted

with the true sarcomas, whose tissue is usually a pale gray. Giant cell growths generally respond to enucleation of the tumor mass without extensive encroachment on the surrounding healthy bone (Fig. 11). In cases seen late, with perforation of the cortical plates, resection of the involved portion of the jaw is sometimes necessary.

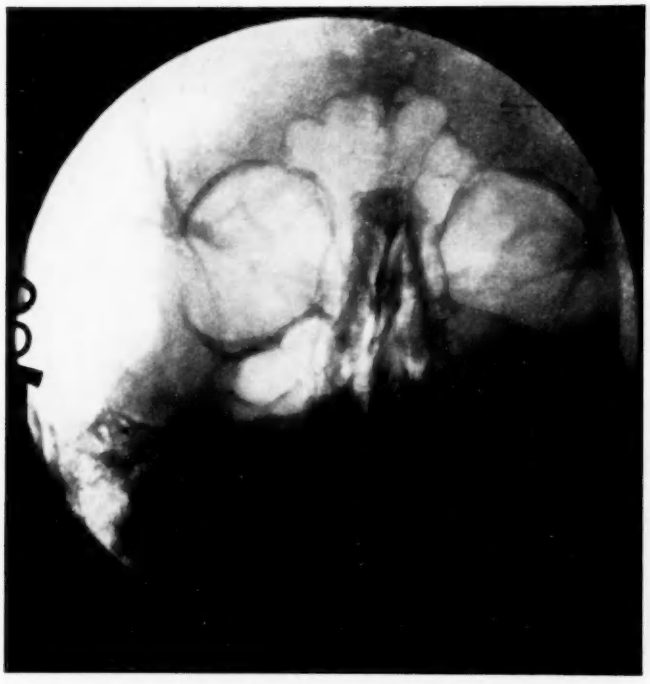


FIG. 11 — (Ivy) — Benign giant cell tumor of maxilla, which had pushed up the floor of the maxillary sinus until floor and roof were in contact. Growth treated by complete enucleation through the mouth.

METASTATIC AND SECONDARY GROWTHS

Carcinoma. When carcinoma involves the jaw bones, it is usually a direct extension from a primary growth of the oral mucous membrane. Occasionally, it may occur as a true metastasis from cancer of the breast or some other distant source, just as other bones in the body are affected in this way. We recently removed some unexplained epithelial tissue from a lesion in the mandible, after which thorough search discovered a primary adenocarcinoma of the rectum, with identical histological characteristics, which had not been suspected.

Thyroid tumors, both benign and malignant clinically, occasionally metastasize to bone. The writer has reported a case of metastasis to the mandible of an apparently benign adenoma of the thyroid, which had been removed about eight years previously. In the presence of an unexplained swelling of the jaw bearing the characteristics of a tumor or cyst, the possibility of a metastatic growth should not be overlooked.

FIG. 12 — (Ivy) — Mixed tumor of palate.

MIXED TUMORS

The term, mixed tumor, is applied to a group of growths most frequently occurring in or near the salivary glands, particularly the parotid, but which may be found in other regions, such as the submaxillary, palate, pharynx, lip or cheek. Clinically, the growth is slow in development, well encapsulated, with a firm elastic consistency, and may be smooth or nodular. It may be very small, or, especially in the parotid region, may reach an enormous size. In the palate or the cheek, the growth is usually firm, smooth, well-defined, and covered by normal mucous membrane (Fig. 12). Histologically, the characteristic cells are difficult to classify, but are probably of epithelial origin. In parts

there is a definite arrangement of these epithelial cells in the form of acini, in others there are large amounts of colloid material. Cartilage and bone formation occurs occasionally. On section of the growth the tissue is found to have a peculiar gelatinous consistency. These growths are not to be regarded as malignant, though local recurrence is common if removal has not been complete.

When small and encapsulated the tumor should be enucleated. In the soft palate, this can be easily accomplished under local anesthesia, after incising and turning back the overlying mucous membrane. Radium and X-ray treatment is not very successful.

FIG. 13 — (Ivy) — Torus palatinus.

MISCELLANEOUS CONDITIONS RESEMBLING TUMORS

Torus palatinus, an exostosis in the region of the median palatine suture of the maxilla, presents an oval, usually smooth, but sometimes nodular hard swelling in the roof of the mouth (Fig. 13). To the uninitiated, this may be mistaken for an osteosarcoma. The torus palatinus is found normally as a developmental condition in a considerable percentage of people, it undergoes no changes in size over a period of many years and causes no pain. Most persons are unaware of the presence of the growth until attention is called to it by a physician or someone examining the mouth or throat. It demands surgical treatment only when the overlying soft tissues become irritated by rubbing or when the swelling interferes with the proper fitting of an artificial denture. In such cases it can be removed with a chisel and ronguer forceps after

the overlying mucoperiosteum has been dissected back. Similar excrescences or exostoses are frequently found beneath the gum on the lingual aspect of the mandible in the premolar regions, and also around the tuberosities of the maxilla. They are not progressive in growth, usually give rise to no symptoms and require removal with a chisel only when they interfere with an artificial denture.

Osteitis fibrosa cystica may occur in the jaw bones, especially in the mandible, producing localized or generalized areas of decalcification alternating with increased density. The diagnosis is established definitely on finding clinical and roentgenologic evidence of similar conditions in other bones, and a disturbed calcium metabolism from hyperparathyroidism. Occasionally, a localized form is found in the jaws, without demonstrable hyperparathyroidism or lesions elsewhere. The treatment in the generalized form is removal of a parathyroid tumor if present. In the localized form, curetting of the bone lesions will usually result in cure.

Osteitis deformans, or Paget's disease, is occasionally seen in the upper jaw. In the maxilla, a hard, smooth swelling covered by normal gum is found in the region of the molar and premolar teeth. As the disease progresses, the swelling may be seen externally, giving rise to a leonine appearance. The X-ray usually reveals consolidation of the facial bones and sinuses, with a peculiar thickening and mottling of the cranial bones. The etiology and cure are unknown, the chief interest of the disease here being its differentiation from other lesions more amenable to treatment.

We occasionally see a more localized *bony hyperplasia* in the upper jaw, which presents a very slowly progressive hard smooth bulging of the bone above the molar and premolar teeth (Fig. 14). It is nearly always unilateral, and sometimes produces a visible external swelling. No pain is experienced. Radiographically, the bone is seen to be homogeneous and of moderate density, sometimes encroaching on the space of the maxillary sinus. The bone is not hard, but can be pared easily with a knife or chisel. Histologically, we have been unable to get the pathologist to find anything to distinguish it definitely from normal bone. Our practice in such cases has been not to attempt to remove all of the overgrowth, but to turn back a flap of the overlying gum and pare off the excess with a knife or chisel to restore the normal contour. So far, we have not had occasion to repeat the procedure on account of recurrence.

FIG. 14 — (Ivy) — Unilateral benign bony hyperplasia of maxilla.

Ranula. Statements frequently appear in the literature to the effect that this is a swelling in the floor of the mouth due to a calculus in the submaxillary duct. This is erroneous. A ranula is a soft, painless, semitransparent swelling beneath the mucous membrane of the floor of the mouth containing clear, thick fluid. The cause is probably inflammatory closure of one of the ducts of the sublingual gland or of one of the smaller mucous glands in this region. It is never associated with a calculus, nor with a swelling of the submaxillary salivary gland, as would be the case if the submaxillary duct were involved. Furthermore, in ranula the submaxillary duct can usually be identified and isolated completely from the cystic swelling. Ranula usually starts on one side of the median line, but as the fluid accumulates the swelling increases so that it may appear to be bilateral. The overlying tissue becomes very thin and finally ruptures, discharging a semifluid resembling white of egg. The swelling then subsides and the process repeats itself. Several methods of treatment of ranula have been suggested, including removal of enough of the

overlying tissue to create a permanent wide opening, but the only way of insuring a cure is by complete dissection of the mass.

This discussion has covered a rather wide territory, yet of necessity many conditions that could have been mentioned have been omitted. It is hoped that what has been said will stimulate the interest of the otolaryngologist in the adjacent field, the mouth and jaws.

DISCUSSION

DR. V. H. KAZANJIAN, BOSTON: You have just heard an instructive paper from Dr. Ivy about benign tumors of the jaws. He has given us accepted and sound methods of treatment of these tumors. There is nothing that one could take issue with. However, in this discussion I would like to emphasize one or two points that Dr. Ivy has already outlined. While the diagnosis of most of these tumors is not difficult, yet in certain instances accurate differentiation is important. Dental root cysts are usually discovered during routine X-ray examination of the teeth, but at times, if the cyst is in the vicinity of the maxillary sinus, it may be quite confusing to know whether or not one is dealing with the antrum or vice versa. This point is very important — to know if one is dealing with an infected sinus or a cyst.

Adamantine tumors are not usually diagnosed early enough to acquire complete cure by conservative operation. As a result they often assume a very large size, necessitating radical removal of a section of the mandible. The reason for this mistake, I suspect, is that in their early stages these tumors are often confused with the simple dental root cysts and treated as such. To avoid mistaken diagnoses it is a good rule to send all specimens of tissue removed to the laboratory.

Surgical treatment of dental cysts merely centers on removal of the cystic membrane and more important, elimination of the cavity which at times becomes quite large. In this respect, I would like to outline the methods I follow in the treatment of cysts of the upper jaw situated near the vicinity of the maxillary sinus and cysts occupying the floor of the nose. The cysts in the lateral aspect of the maxilla, unless treated early, seem to grow posteriorly and medially until they eventually encroach upon the maxillary sinus and nasal cavity. The partition between the maxillary sinus and the cyst is at first composed of a thick layer of bone. As the cyst cavity grows larger this bony partition gradually absorbs until the mucous membrane covering of the cyst and the antrum adjoin one another. Oftentimes the pressure of the cyst pushes the mucous membrane towards the lateral wall of the antrum and eventually obliterates the greater part of the sinus. During this process of expansion, the lining of the antrum does not disappear. It becomes folded upon itself leaving a very thin space for secretion to seep through. Dr. Ivy prefers to make a very wide opening through the mouth into the cyst cavity leaving the lining membrane intact, thereby establishing permanent drainage and eventually shrinkage of the

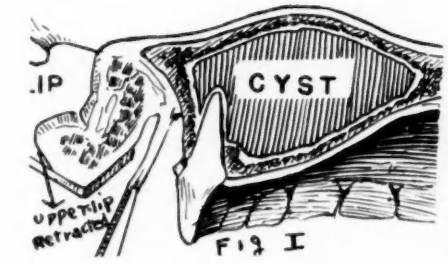


Fig I

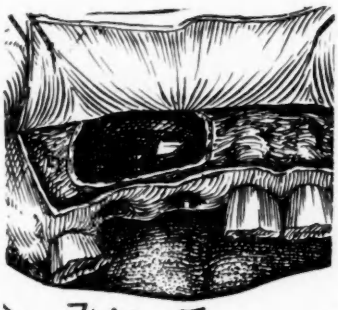


Fig II

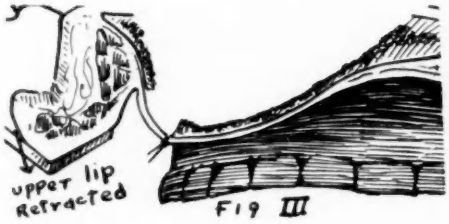
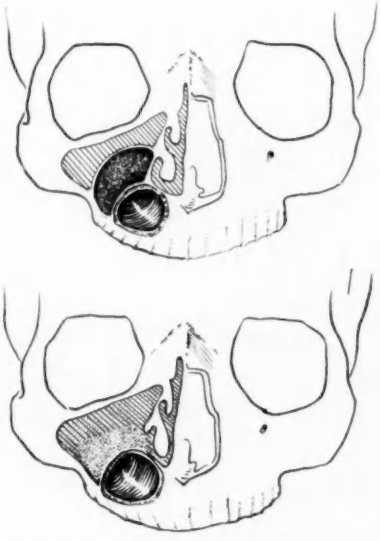


Fig III



cyst. I use the following procedure: The entire cyst is shelled out through the buccal opening and the wall separating the maxillary sinus from the cyst is obliterated and the sharp bony edges along the border are trimmed. A large opening is then made under the lower turbinates for drainage. The mouth wound is then closed with silk sutures. There is very little post-operative reaction following this operation and very little care is needed. While I have not had occasion to open these cavities, there is

reason to believe the cystic cavity, being unnatural, is eventually eliminated, the maxillary sinus assuming its normal size and contour.

Cysts of the anterior part of the maxilla when large invade the greater part of the palate and floor of the nose. One can see a definite change in the contour of the palatal arch as well as the contour of the floor of the nose. These cysts are operated through a mouth opening. Cystic membrane is removed. Not infrequently, one sees the bony shell in the center of the cyst completely absorbed so that in separating the membrane one must be careful not to perforate the palatal mucous membrane. Elimination is accomplished by exposing the cystic cavity to the nasal cavity. To do this, it is necessary to remove mucous membrane, also the bone that separates the floor of the nose from the cyst. All the bony projections should be trimmed so as not to allow any blind pockets. The bleeding is stopped and the mucous membrane flap is brought down to its original position and sutured in place, and thus joining the cystic cavity to the nasal cavity. I find this procedure highly desirable in large cysts that invade the floor of the nose.

Dr. Fred Z. Havens, Rochester, Minn.: I realize that many nose and throat men do not maintain any more than an academic interest in cysts of the jaws. Those of you who do and who take an active interest in the treatment of these things will realize without my telling you what a nice paper Dr. Ivy has given us.

I would like to emphasize the point Dr. Ivy made regarding the necessity of radical removal of an adamantinoma. These tumors are certain to recur and to progress if not removed completely. Their progress is very slow, in fact, so slow that the patient may live ten, fifteen or twenty years and suffer untold agony before death mercifully intervenes. The importance to the patient, therefore, is so great that one need not hesitate to employ radical measures in treatment of a tumor of this type.

In recent years, at the Mayo Clinic, we have used surgical diathermy in the treatment of adamantinoma with gratifying results. The tumor is scooped out and following this the entire base of the wound is cauterized.

There is disagreement among pathologists as to whether adamantinomas should be considered to be benign or malignant. Excellent investigators support each side of the argument. I prefer the classification by which adamantinomas are placed in the group of malignant tumors rather than to consider them benign. Their tendency to recur, to progress, and to invade surrounding tissues is characteristic of a malignant tumor. I have encountered two instances in which metastasis had occurred.

Dr. Ivy's discussion of mixed tumors of the palate and pharynx should be of especial importance to the otolaryngologist. These tumors usually attract the patient's attention fairly early when they occur in the palate but when they occur behind the tonsil they may become surprisingly large before the patient realizes that anything is wrong. I agree that radium is not of much benefit in these cases. Usually these tumors can be enucleated readily since they remain encapsulated until a late stage. If the tumor is behind the tonsil, the external carotid artery should be ligated

before enucleation is undertaken, for hemorrhage may be uncontrollable otherwise.

Dr. Ivy classifies the mixed tumor as benign. Here again there is disagreement among pathologists. It recurs following incomplete removal, it progresses, albeit usually very slowly, and it ultimately breaks its capsule to invade surrounding tissue. It occasionally gives rise to metastasis. These clinical characteristics would seem to warrant its classification as a malignant tumor.

Dr. E. F. Risdon, Toronto: I would like to compliment Dr. Ivy on his clear and concise discourse and further that his treatment is correct and simple. So frequently one is forced to listen to complicated treatments. Seldom have I seen it of much value.

There is one point that I rather regret Dr. Ivy did not bring out, that he did not have time for it, and that is the chronic low-grade infections of the upper jaw. One sees them frequently, the huge tumors, and unless they are studied carefully by stereoscopic X-rays they are overlooked. On irrigation, perhaps little or no pus is obtained, but at operation you will find that the whole upper wall can be removed perhaps through the intra-orbital range. They should not be overlooked.

From the standpoint of osteofibrosis, I have seventeen cases. That may not be many. In those cases there was no other possible lesion in the body. The pathological report was osteitis fibrosis. In those cases, just as Dr. Ivy said, from the standpoint of treatment it is only necessary to shell them out. One interesting feature about it is that when you expose the bone the outer cortex is like the shell of an egg. That is removed and you have something which looks like bony solids. It can be removed while the antrum is entirely obliterated. They do remarkably well and the patient is able to wear a denture if not too much is removed. I do not think you will find much in the literature relative to this type of treatment. In my opinion, it is the surgical insult that cures them. They are quite vascular and they do not return.

From the standpoint of dentocystic diseases, the simple dental cyst directly involves the floor of the sinus, usually unnecessary to open into because they can be approached from the mouth.

The osteitis fibrosis and the ameloblast seem to be large tumors on X-ray. On is more edematous than the other.

From the standpoint of nasopalatine cysts, my own opinion is that they should be seldom operated on. First, because they are difficult to approach. The lining is very thin since it is an occlusion cyst and is quite difficult to remove without the loss of teeth.

Next is a tumor which is difficult of diagnosis. I have always been of the opinion these tumors were malignant, at least, so close to it they should be considered as such. I refer to the adamantinomas. Any growth near the jaw is classed in that manner. The contiguous cyst tract isn't popular, but the name adenoma has become very popular. I use diathermy by selection, even if many failures have been noted. I quite prefer to excise them widely.

PATHOLOGIC EXAMINATION OF TWENTY-FOUR CASES OF PETROSITIS

TYPE — SITE — EXTENT — RELATIONSHIP TO SYMPTOMS

ISIDORE FRIESNER, M.D.

NEW YORK

THE STUDY of the pathologic material that we have accumulated in the last ten years demonstrates such interesting facts that although they have been presented by us in other publications, we deem it timely to review them here.

These facts have such an important bearing on the understanding of the type of lesion, its site, extent and its relationship to the symptoms of petrositis that they are of considerable import in treatment. Most of the cases that we have had an opportunity to examine at post-mortem had already developed intracranial complications when they were admitted to the hospital. However, so far as we were able, we have carefully gone into the history of the acute infection of the ear and mastoid and believe we are in a position to compare the symptoms reported with the disease process found on histological examination. One of the first facts that is outstanding as a result of our examinations is the following:

1st — Extensive suppuration can occur in the petrous pyramid of patients who died of intracranial complications without any symptoms of the bone disease.

2nd — In the consideration of petrositis it is important to include pathologic changes which occur in the perilabyrinthine structures as well as those that occur in the apex. Cases of perilabyrinthitis and deep extra dural abscess, which undoubtedly should be considered as petrositis, have been reported in the literature. *It must not be forgotten that by no means does the lesion in the petrous pyramid in all cases extend to the apex. Nor are the symptoms dependent upon extension of the lesions to the apex.*

It seems to me that from this fact and from the fact that extensive disease may occur in the pyramid with no signs nor symptoms whatsoever, one must strongly suspect that with the exception of discharge, no other symptoms of this condition are directly due to disease within the bone. The facts which we have learned from the examination of the first twenty-four cases have not been materially altered by the study of the subsequent cases.

3rd — In the majority of our cases the infection spread from the tympanum and antrum through the perilabyrinthine cells. One may regard the labyrinth as suspended in the petrous pyramid in a ring of pneumatic structure. This ring need not be complete. Of course, it is not invariably present but when it is present it is through this pneumatic ring that infection from the ear and antrum extends in the bone along the floor of the skull. There are no definite lines of demarcation between groups of cells composing this ring since they all intercommunicate.

4th — The infection spread from the tympanum and antrum along the superior and posterior perilabyrinthine structures in nineteen of the twenty-four cases.

5th — The postero-superior route, we found, was by far the most frequent. It cannot be stated too emphatically that lesions in the petrous pyramid do not always extend to the apex. *In the majority of our cases, the greatest expression of the disease process was noted in the petrous pyramid between the superior semicircular canal and the internal auditory meatus. Furthermore, in fourteen of the twenty-four cases, the lesion was most manifest at the superior-posterior margin of the petrosa.*

6th — In only three of the cases were the inflammatory changes in the apex more marked than those in the remainder of the pyramid. The duration of the infectious process may be a factor in the extent of the involvement of the petrosa and apex. As is well known, the suppurative process frequently erodes the bony cortex of the petrosa, along the middle or posterior fossa at an appreciable distance from the apex, and produces an extra dural abscess. *These changes may take place before any considerable degree of involvement of the apex has occurred.*

7th — CONCLUSION. Correlation of our anatomic and histopathologic studies with our clinical experience have led us to the following conclusions:

1. That many suppurations in the petrous pyramid tend to drain and heal spontaneously. Therefore, we are extremely conservative.

2. That by no means does the lesion in the petrous pyramid, in all cases, extend to the apex, nor are the symptoms dependent upon extension of the lesions of the apex.

3. That the postero-superior route is the frequent pathway of extension and that the greatest expression of the disease process is noted in the area between the superior semicircular canal and the internal auditory meatus, at the posterior superior margin of the petrosa.

4. That in our clinical experience, even after weeks of profuse discharge from the ear, normal hearing or approximately normal hearing is restored when healing takes place.

5. With the above facts in mind, we have followed a conservative course and even when under the necessity of operating have tried to preserve the function of the ear.

DISCUSSION

Dr. Ernest M. Seydell, Wichita, Kan.: It is both a privilege and a pleasure to have this opportunity to discuss Doctor Friesner's masterly presentation. Unfortunately he has left little that I can add. This is especially true since my views on this subject are fully in accord with those he has expressed.

In the short time allotted to me, I shall try to bring out a few facts, some of which will be mere reiteration of what has been said. Repeating them will help, I hope, to emphasize their importance.

It is essential to keep in mind the following anatomical facts. The presence of a pneumatic mastoid process does not necessarily mean that the petrous perilabyrinth is cellular. In Myerson's series the mastoid process was pneumatic in 38 per cent of his cases, whereas only 11 per cent of the petrous pyramids were cellular. Friesner reported that 50 per cent of his mastoids were pneumatic in comparison with 16⅔ of his petrous pyramids, while the apex in these cases was pneumatic in only 5 per cent. Again, the two petrosæ are not always similar in structure. Myerson found that in 4 per cent of his series only one pyramid was pneumatic and in 7 per cent there was a distinct difference in the structure of the two. These facts have, of course, an important bearing on the interpretation of X-ray findings in cases of suspected petrosal suppuration.

Generally speaking, pneumatization of the petrous pyramid does not take place before the fourteenth year. Nevertheless in the series of cases of petrositis which I reported last year 34 per cent occurred in children

under 10 years of age. In these patients, therefore, the pathologic lesion was not an osteitis but an osteomyelitis.

I am in accord with Dr. Friesner when he states that in the majority of cases of petrositis, the diseased process does not extend into the petrous apex but is found principally between the superior semicircular canal and the internal auditory meatus.

In reference to the symptomatology of suppuration of the petrosal pyramid, again I am in accord with Doctor Friesner. I agree with his statements that the symptoms are not necessarily dependent upon the extension of the lesion into the apex of the petrous bone and that providing other etiological factors have been excluded, the presence of pain is more important than its location. In the cases which I reported only 36 per cent had pain deep in the eye or immediately around the eye and in 5 per cent of the cases, there was a complete absence of pain hemicrania. As we all know, pain behind the eye prior or subsequent to a mastoid operation does not necessarily arise from suppurative petrositis. In a case of this character which came under my observation this year the pain was caused by a nodular myalgia of the neck muscles. My own experience has been similar to Doctor Friesner's in reference to the fact that cessation of pain marked the beginning of recovery. We know, however, that this cannot be regarded as a definite rule.

I think that most of us will agree that the great majority of cases of petrositis will resolve either spontaneously or following a paracentesis or simple mastoid operation. The fact that symptoms of petrositis may be present is, therefore, not in itself a valid indication for operation on the petrous perilabyrinth. On the other hand, we must recognize the fact that numerous cases of petrositis have not been diagnosed except in post mortem.

In my previous case reports no suggestion of petrositis could be obtained from the history in 11 of the 14 patients who died from intracranial complications following suppuration of the petrosal pyramid. In a recent report of the Mayo Clinic, Williams stated that in 5 of their cases, petrositis was not suspected prior to the mastoid operation. The lesson to be learned from this is that in performing a mastoid operation, thorough search must be made for an extension of infection in those areas where cell tracts are known to lead into the petrosa. In cases of acute suppurative mastoiditis where symptoms suggest a petrosal involvement, an early mastoid operation would seem to be indicated.

My method of operative procedure is very similar to that described by Doctor Friesner. I, too, refrain from performing a radical mastoid operation whenever possible. Thus far I have used this procedure on two occasions. In these patients, no infected cell tracts leading into the petrosa were found. Following the operation, the symptoms continued unabated and a radical mastoid operation was then performed. In both cases, I failed again to find a fistula in the tubal region. The two cases recovered. It is interesting to note that in the 30 cases of suppurative petrositis operated at the Mayo Clinic and reported by Williams, they found it unnecessary to perform a radical mastoid operation in a single instance, although in their series there were three cases where no infected cell tracts were found.

Dr. Robert L. Moorhead, Brooklyn: My discussion of this paper is based entirely on a personal series of operative cases. Our clinical findings, to a large extent, back up Dr. Friesner's pathological findings.

Relative to the subject of pain: we have had cases with no pain. The typical eye pain so often described by most writers is present certainly in not more than 50 per cent of cases. The pain in the head is present in a large majority. We have had one case with pain over the opposite eye. I just mention this as a disturbing factor. We have had two cases with pain in the teeth; although it is not supposed to be pathognomonic of the condition, it was a determining factor. Several of the cases have had no pain whatever.

Dr. Friesner mentioned the discharging ear and the cessation of the discharge. A few years ago we were told that a discharging ear was characteristic of these conditions. We had to have the pain behind the eye. The discharging ear and the temperature, and so forth, had to be there. We now know this is not true. The cases that we have to look out for most of all are the cases where the discharge is lessening.

Dr. Friesner mentioned the fact that he found pathologically several cases of petrous involvement without any symptoms whatever, which is undoubtedly true. We have confirmed this clinically a number of times in operating on meningitis cases. We always investigate the petrous in meningitis, and find many times the apex involved. My own statistics show about 20 per cent of the meningitis cases as having involvement of the petrous pyramid.

Dr. Friesner mentioned that the route of infection in a large percentage of cases was through the perilabyrinthine cells. This is borne out clinically in something like seven or eight of the 26 cases we have had in which pus was found in the apex. Fistulæ were observed, I think, in seven leading into the apex.

Insofar as treatment is concerned, conservatism without any question should be advocated. A simple mastoid operation does not cure a large percentage of the petrous apex infections. The larger percentage of cases observed had already been operated on for simple mastoid.

We should be conservative, however. I do not think any case should be operated needlessly just because somebody feels there is an involvement in the apex. When definite symptoms then are absent, the radical approach is desirable. Years ago we did that without any question.

I can recall several cases in 1930 and 1931 where we unknowingly watched the symptoms develop. The typical syndrome was absent and they developed intercranial complications.

I think one of the big factors in such cases is the conservation of hearing. We can approach the petrous apex or the petrous pyramid comparatively easily along the middle fossa. We do not need to do a radical.

I have audiograms of practically all of my cases, with the exception of two, one of which had had a radical. Another had had a labyrinth infection. Practically all have normal hearing, quite unlike the expected following a radical approach.

SYMPATHETIC INNERVATION OF THE NOSE*

OLOF LARSELL, Ph.D.
BY INVITATION
and RALPH A. FENTON, M.D.

PORTLAND, ORE.

THE RESULTS of anatomical and physiological investigation indicate that the nasal membranes have a rich innervation from several sources. These nerves may be divided for our present purpose into two main groups, namely, afferent and autonomic. The olfactory nerves are omitted from consideration.

The autonomic fibres come in part from the superior cervical ganglion through the internal carotid plexus. These are the thoracolumbar autonomic or sympathetic fibres. Others come from the great superficial petrosal nerve, synapsing in the sphenopalatine ganglion. They include vasodilator and secreto-motor fibres. These are the cranial autonomic or parasympathetic fibres. The sympathetic fibres from the superior cervical ganglion are chiefly vasoconstrictors, but also include vasodilators, according to Dastre and Morat.[1] The preganglionic fibres arise in the lower cervical and upper thoracic cord, reaching the ganglion through the cervical sympathetic trunk.

Vasodilator and secretory fibres from the sphenopalatine ganglion to the nasal mucosa have been described by various investigators. Tschellussow[2] described a vasomotor path to the nasal membranes, with preganglionic fibres from the nerve of Wrisberg to Meckel's ganglion and postganglionic fibres from this ganglion to the nasal membranes. Blier[3] has shown that the Vidian nerve and the posterior nasal nerves convey both vasoconstrictor and vasodilator fibres to the nasal membranes. He does not differentiate the two parts of the Vidian nerve, namely, the great superficial petrosal and the great deep petrosal nerves. Electrical stim-

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ulation of the Vidian nerve would affect both, with confusing results. Blier, however, obtained only vasoconstriction on stimulating the Vidian nerve after applying nicotine to the sphenopalatine ganglion, when he waited long enough for the drug to produce its narcotic effect. The immediate effect of the nicotine itself was a transitory vasodilatation, indicating stimulation of sphenopalatine ganglion cells whose fibres constitute the vasodilators. The vasoconstrictor effect of stimulation of the Vidian nerve was lost when the superior cervical ganglion was removed and sufficient time was allowed for degeneration of the fibres from these cells. Obviously the vasoconstriction was due to stimulation of fibres from the superior cervical ganglion to the deep petrosal part of the Vidian nerve.

It should, therefore, be emphasized that the Vidian nerve consists of two functionally distinct parts, derived from widely separated regions of the nervous system (Figs. 1 and 3). This has been pointed out on anatomical grounds.[4] The evidence from physiological experiment should serve to make the facts even clearer. It is necessary to specify which of these parts, namely, the great superficial petrosal nerve, or the great deep petrosal nerve, is concerned, if we would correctly interpret phenomena due to involvement of the Vidian nerve.

Jung, Tagand and Chavanne[5, 6, 7] and others have shown that the glands of the nasal region and palate are innervated by postganglionic fibres from Meckel's ganglion. There is lack of unanimity, as well as of clarity, in the writings of different authors with regard to the course of the preganglionic secreto-motor fibres. The preponderance of evidence indicates the great superficial petrosal nerve as the path.

No physiological differentiation has been made between the vasodilators from the superior cervical ganglion and those from the sphenopalatine ganglion. Evidently the vasoconstrictor element of the superior cervical ganglion is much more important than the vasodilator. Yet Dastre and Morat[1] and also Jung, Tagand and Chavanne[7] have adduced seemingly reliable evidence that vasodilators ascend from the superior cervical ganglion. There is, however, no evidence of vasoconstrictor fibres in the great superficial petrosal nerve which relay in the sphenopalatine ganglion.

With this background of anatomical and experimental work, it is suggested that the clinical picture of congestion of the nasal membranes associated with intestinal stasis, pelvic congestion, etc., may have its physiological explanation in an overflow of vaso-

dilator impulses to the nasal membranes. Whether such overflow would be through the sympathetic or the parasympathetic route is not clear. The anatomical pathway for the sympathetic route is obvious. It is more difficult to account anatomically for an overflow of stimuli causing pelvic parasympathetic congestion, for example, to the parasympathetics of the nasopharynx. The pathway would necessarily lie in the cord, and there is no anatomical evidence for such a path. On the other hand the abundant nasal discharge frequently accompanying nasal congestion associated with colonic or pelvic congestion suggests the cranial parasympathetic route, since secreto-motor fibres belong to it. Such pathways and their relation to visceral organs must be taken into consideration if we would understand some of the phenomena of nasal physiology. The probable connection between exacerbation of residual sinus infection and such periods of vascular congestion in the nasal membranes is apparent.

The afferent fibres of the nasopharynx end chiefly in the mucous membrane itself, either in the epithelium or in the connective tissue beneath. It is probable that some, here, as elsewhere in the body,[8] end in relation to the blood vessels of the nasal mucous membrane. Like other nerve fibres these undoubtedly respond to a sufficient stimulus anywhere along their course.

The afferent fibres are derived in large part from the Gasserian ganglion of the trigeminus.[9] There is evidence,[10] however, that afferents arising in the geniculate ganglion, in the nodose ganglion, and possibly some from the petrous ganglion, reach the nasopharynx and palate (Fig. 2). Kuntz[11] has adduced experimental evidence indicating that afferents from the upper thoracic nerves reach the common carotid artery. Through the common carotid plexus and the internal carotid plexus, these fibres reach the nasal membranes and other parts of the head. The evidence that the fibres are connected with the dorsal root ganglion cells is not conclusive, as Hinsey[12] has pointed out. Kuntz sectioned the common nerve trunks distal to the junction of dorsal and ventral roots, thus allowing the possibility of ventral root fibres being involved in the resulting degeneration. Hinsey's suggestion that the myelinated fibres which degenerated as a result of this operation may be preganglionic fibres to scattered ganglion cells along the course of the carotid artery and its branches must be admitted as a possibility.

The evidence, clinical, histological and experimental, points for the most part, toward the interpretation made by Kuntz. The

clinical studies of Temple Fay[13] receive their most rational interpretation on this basis. To this evidence we can add the results of study of Pal-Weigert serial sections of the human internal carotid artery and its nerve plexus, including the connections of the latter with the great deep petrosal and other neighboring nerves.[10] These studies, while not so detailed as those of Christensen[14] on the cat, due to lack of sufficient material and limitations of technique on autopsied human material, agree so far as they go, with the results obtained by Christensen.

Myelinated fibres of large and medium size are found in the internal carotid plexus in man. Reconstructions from Pal-Weigert series also show large myelinated fibres in one of the connecting filaments between the nodose ganglion and the petrous ganglion. We have also found such fibres in osmic acid series of the rabbit. Myelinated fibres of similar size and appearance continue into the nerve of Jacobson. Part of this bundle branches into the lesser superficial petrosal nerve, part into the superior carotico-tympanic nerve, and part into the internal carotico-tympanic nerve. Whether or not they all have their origin from cells of the petrous ganglion it is impossible to determine. Some may come from the nodose ganglion through the connecting bundle above described. It is not possible, however, to trace a continuity between the latter and the fibres of Jacobson's nerve among the cells of the petrous ganglion.

Small myelinated fibres, corresponding in size to preganglionic fibres elsewhere, are present in our Pal-Weigert series between the petrous ganglion and the superior cervical ganglion. They pass from the latter into the carotid plexus. It does not appear probable that they are preganglionic fibres in this case. We also find large myelinated fibres from the superior cervical ganglion to the carotid plexus and great deep petrosal nerve. They are lost in the superior cervical ganglion but it is evident that they do not extend into the cervical sympathetic trunk. It is probable that they bridge across to the petrous or nodose ganglia, but definite histological evidence is lacking in our material.

If the small myelinated fibres to the petrous ganglion and the large fibres probably passing to the petrous and nodose ganglia have cells in these ganglia, there is anatomical basis for degeneration of fibres in the carotid plexus where the superior ganglion was removed, as obtained by Kuntz in the cat. He does not state the size of the fibres which degenerated. It would also explain the results obtained by Davis and Pollock[15] of pain in animals from stimulation of the superior cervical ganglion.

Myelinated fibres are present in our human material in the carotid plexus below the level at which the great deep petrosal nerve joins it. Whether these continue to the upper thoracic dorsal root ganglia in man we have no means of ascertaining at present. The clinical observations of Temple Fay give some basis for assuming that they do.

In connection with the problem of referred pain from the nasal region another group of afferent fibres, not directly connected with the nasal mucosa, must be considered. These are the cutaneous branch of the facial nerve, the great auricular nerve, and the cutaneous nerves to the neck and shoulder. Possibly deeper afferent nerves are also involved in so-called referred pain. The nerves named supply the skin of the mastoid region and of part of the head and neck. Various explanations of referred pain have been offered by Head, MacKenzie and others. If the general idea of an overflow of stimulation from visceral sources, within the central nervous system, eventually affecting cutaneous pathways, which somehow project the sensation to corresponding segmental surfaces of the body, be accepted, then the relation between afferent fibres in the great superficial petrosal nerve to fibres entering the medulla oblongata from the cutaneous VII, is sufficiently close to explain referred pain in the mastoid region. Likewise the afferent fibres from the nasal region to the petrous or nodose ganglion, whose central roots must enter the bulb with cutaneous fibres of the IX and X nerves, supply the connections necessary to account for basal headaches. The pain in the shoulder and arm of which some patients complain, however, must be accounted for on a segmental basis in some other way. The explanation offered by Kuntz would do this.

The recent tendency is to explain so-called referred pain as due to vasomotor reflexes in the segments affected, producing changes in the skin, either chemical or ischemic. These skin changes are then considered as giving rise to painful impulses directly involving the cutaneous afferent nerves of the segment affected. The fact that local anæsthesia applied to such regions relieves pain lends great weight to this explanation and makes it appear as the simplest and most plausible yet offered.

In a series of seven patients with involvement of the sphenopalatine region and complaining of pain over the mastoid region, with symptoms typical of cases which respond favorably to cocainization of the sphenopalatine ganglion region, we injected the skin between the ear and the mastoid process with one per cent

novocaine. This procedure was immediately followed by cessation of pain.

These results confirm those of Weiss and D. Davis[16] as well as those of L. Davis and Pollock[17] elsewhere in the body. They point to reflex vasomotor disturbances giving rise to pain in the mastoid region by involving the cutaneous VII nerve and possibly the great auricular. It is difficult, however, to account for so restricted a localization of vasomotor effects. Perhaps the thinness of the soft tissues above the mastoid process, with correspondingly small blood supply normally, makes this region the first to be sufficiently affected to produce painful sensations when reflex ischemia is brought about by irritation of the sphenopalatine region.

The pathway from the source of irritation in the sphenopalatine region, described by Kuntz as reaching the upper thoracic segments of the cord by the great deep petrosal nerve and carotid plexus, would be a favorable one for producing reflexes in this part of the cord. Vasomotor impulses originating here would reach the superior cervical ganglion, where they would be relayed to the postganglionic fibres of the carotid plexus which reach the posterior auricular artery and its branches. In basal headache and in pain involving the neck, shoulder and arm, associated with disturbance in the sphenopalatine region, a larger spread of the impulse within the cord so as to include vasoconstrictor nerves to these regions would be necessary. The afferent segmental nerves would be affected, and, through their connections with the spinothalamic tract, would produce conscious pain.

ABBREVIATIONS FOR ALL FIGURES:

ch.tym., chorda tympani; *com.car.art.*, common carotid artery; *ext.car.art.*, external carotid artery; *Gas.gn.*, Gasserian ganglion; *gen.gn.*, geniculate ganglion; *inf.cerv.gn.*, inferior cervical ganglion; *int.car.art.*, internal carotid artery; *jug.gn.*, jugular ganglion; *lat.sp.thal.tr.*, lateral spinothalamic tract; *mid.cerv.gn.*, middle cervical ganglion; *n.cut.VII*, cutaneous VII nerve; *n.max.*, maxillary nerve; *n.gr.sup.pet.*, great superficial petrosal nerve; *n.gr. d.pet.*, great deep petrosal nerve; *n.Jac.*, nerve of Jacobson; *nod.gn.*, nodose ganglion; *n.pal.*, palatine nerve; *n.post.nas.*, posterior nasal nerves; *n.VII*, facialis nerve; *n.IX*, glossopharyngeal nerve; *n.X*, vagus nerve; *pet.gn.*, petrous ganglion; *ret.form.*, formatio reticularis; *ret.sp.tr.*, reticulo-spinal tract; *r.IX*, root of glossopharyngeal nerve; *sol.tr.* and *nuc.*, tractus and nucleus solitarius; *sph.pal.gn.*, sphenopalatine ganglion; *sp.V tr.*, spinal V tract; *sup.cerv.gn.*, superior cervical ganglion; *tym.plex.*, tympanic plexus.

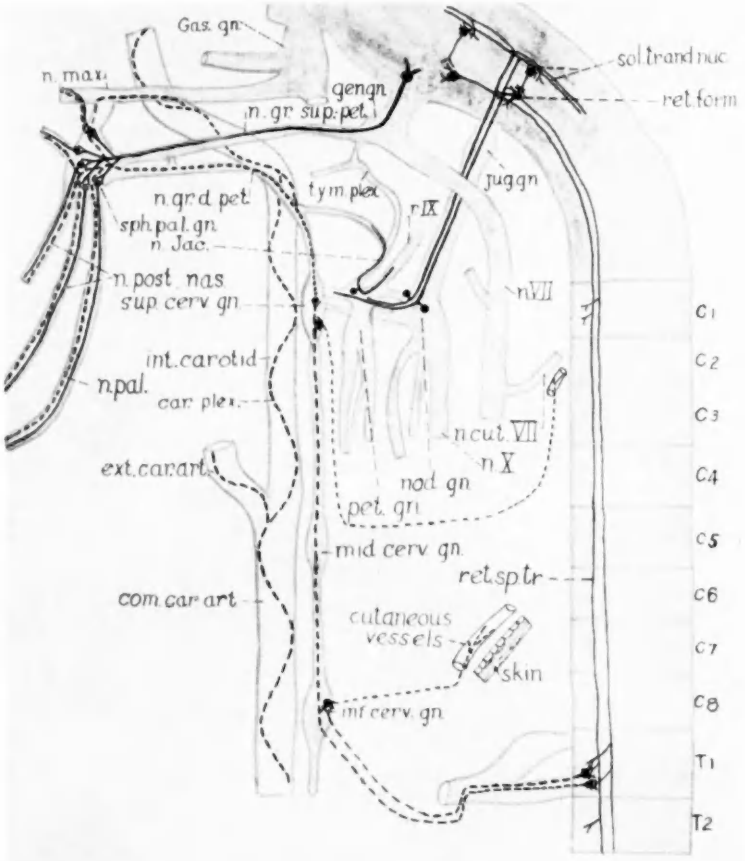

FIG. 1 — (Larsell and Fenton) — Sympathetic (broken lines) and parasympathetic (continuous lines) connections of sphenopalatine ganglion and neighboring nerves. Part of the reticulo-spinal tract, the tractus solitarius and visceral afferent nerves into the latter are also shown. Visceral afferent fibres from the VII nerve also enter the tractus solitarius, but are omitted for simplicity in the diagram.

There is another path by which impulses from the sphenopalatine region may reach the cord segments necessary for vasoconstrictor effects in the region under discussion. This is the reticulo-spinal tract, having its origin in the brain-stem and ending at various levels of the spinal cord (Figs. 1 and 2). Allen[18] has shown that this tract carries vascular reflexes. He has also demonstrated[9] that stimulation of the trigeminus in the nose or of the central stump of the severed vagus produces vasoconstriction and rise of blood pressure. Stimulation of facialis afferent fibres

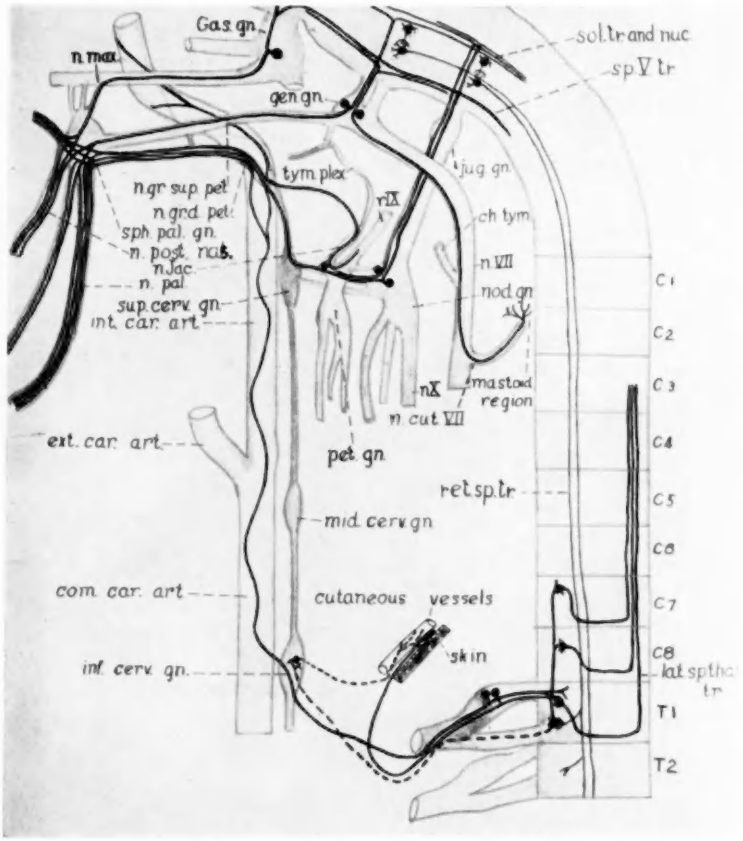

FIG. 2 — (Larsell and Fenton) — Afferent fibres in relation to the sphenopalatine ganglion. The cutaneous vasomotor reflex arc from the upper thoracic segments of the spinal cord is also shown, with the probable relation of segmental afferent fibres. The probable central connections are indicated.

from the nasal region or of glossopharyngeal fibres, if these are present as above suggested, would undoubtedly have the same effect. These would be visceral afferent fibres and therefore would end centrally in the solitary tract and its nucleus. Allen[19] has also pointed out the pathway of reflexes from the solitary tract to the spinal cord, namely, by way of the cells of the solitary tract nucleus to the reticulo-spinal tract and through it to the cord. Here connections are made with the preganglionic fibres of the sympathetic vasoconstrictors emanating from the lower cervical and upper thoracic cord. The superficial vessels of the mastoid region, the superficial and possibly some of the muscular

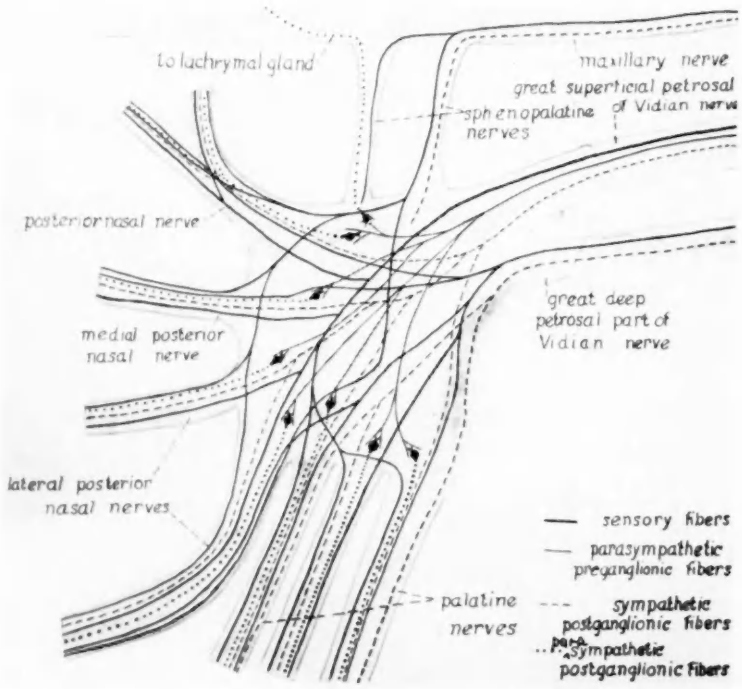

FIG. 3 — (Larsell and Fenton) — Sphenopalatine ganglion and its connecting fibres.

vessels of the base of the head, neck, shoulder and arm could thus be affected. The area involved would probably be in rough proportion to the degree of involvement of the sphenopalatine region, although it would no doubt vary with the individual also.

This suggested pathway, as well as that suggested by Kuntz, should be susceptible of experimental study. In either case the possible vasoconstrictor reflex arcs, together with the VII, IX and X cranial and the cervical nerves, offer an explanation of so-called referred pain from the sphenopalatine region which has much experimental and anatomical evidence in its favor. The hypothesis that vasoconstriction is reflexly produced by irritation of visceral afferent nerves offers a basis for understanding the beneficial effects of surface massage in cases of basal headache and related conditions.

* * *

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Research Report on
SUBSTANCES RESEMBLING EPHEDRIN IN EXPERIMENTAL AND CLINICAL SINUSITIS*

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Following our 1935 report of the reparative reactions shown in acute experimental sinusitis in the cat by certain substances resembling ephedrin, Dr. K. K. Chen requested us to report on four of these drugs, especially as to their shrinking powers and their reparative effects in chronic sinus inflammation.

SUMMARY OF EXPERIMENTS

In a considerable series of private patients the effects of these substances on the inferior turbinate were tested by swabbing on 1% solution, and in a smaller series the solution was dropped into the nose with the head reversed, with the following results:

Tetrandrine methiodide 1% and ½% solution, pinkish precipitate at room temperature after 24 hours, hence was used only a few times. Shrinkage rather marked, but some complaint of bitter taste and slight burning;

Hordenine methiodide 1% solution, produced fair shrinkage, slight sneezing and burning, rather persistent bitter-acid taste;

Benzyl dimethlyamine methiodide 1% solution gave excellent shrinkage, with little or no burning, but was slightly bitter;

Beta phenylethylamine 1% solution produced fair shrinkage, with slight burning and a fairly bitter taste.

No evidences of systemic absorption, changes in pulse or blood pressure were noted.

Two middle-aged individuals requiring radical maxillary operation for chronic infection received three injections of hordenine

* Research requested and financed by Eli Lilly and Co., from the Department of Anatomy and Otolaryngology, University of Oregon Medical School.

methiodide 1%, and of beta phenylethylamine 1%, and the tissues were removed at operation one day following the third injection. No clinical improvement was reported by the first patient, while the second reported very great relief from congestion and was with difficulty persuaded to have his operation; no purulent exudate was found, and the chronic membranes, filled with plasma cells, with little or no evidence of reparative changes.

Cats in which the sinuses had been infected for six weeks received perfusions with the various solutions, which were also tested in a certain number of uninfected sinuses. Apparently all these solutions are somewhat irritating to normal ciliated sinus epithelium, especially with beta phenyldiamine and tetrandrine methiodide.

Little or no evidence of active stimulation of repair could be found in most of these chronic infected membranes, although less damage to cilia was found with benzyl dimethylamine methiodide, and more migrant leukocytes with beta phenylethylamine. Eosinophiles were more noticeable with use of tetrandrine methiodide and of beta phenylethylamine.

The value of these substances would seem to lie in their clinical use in relatively acute conditions, after determination of necessary pharmacologic factors, particularly for benzyl dimethylamine methiodide and beta phenylethylamine.

DETAILS OF EXPERIMENTAL STUDIES

The experimental work was carried out with the technical assistance of Dr. Wallace Shearer in the Department of Anatomy. Maxillary antrum surgery was done by Dr. W. N. Thompson, resident in the Department of Otolaryngology.

Three groups of cats were chronically infected with hemolytic streptococci, and the various 1% solutions were perfused through their sinuses 72 hours before killing the animals.

The chronic infections were accomplished by incising the skin over the frontal sinuses under ether anæsthesia. The subcutaneous tissues and periosteum were scraped away and then by the use of an antrum needle a small amount of semi-solid 1% agar was injected into the sinus, thus partially closing the sinus ostium. This was followed by injection of about 1 cc. of a 24 hour culture of hemolytic streptococci obtained fresh from a virulent hospital case. In about 48 hours, in most cases the cats developed a head cold with sneezing and a nasal discharge. This usually remained as an acute infection for about 3 weeks, sometimes longer. At

the end of 5 weeks these animals are considered chronically infected, in accordance with previous microscopic studies.

In the first group of cats only the right sinus was infected, the left sinus being normal. In this group both sinuses, the infected and the normal, were perfused with the solutions until the fluid flowed from the nostrils. In the second group also, only the right sinus was infected, but in this case each animal received two perfusions in each sinus 24 hours apart. In the third group both sinuses were infected but only the right sinus was perfused with the solutions.

In each case the animals were killed 72 hours after final perfusion. Eight cc. of 1% trypan blue was injected into the subcutaneous tissue of the neck at the time of the last perfusion.

Hordenine methiodide 1% and beta phenylethyldiamine 1% were used in the available human material. Two patients, each scheduled for radical removal of the left maxillary sinus membrane, were used. Six days previous to the operation the left antrum in each case was irrigated with water. At the end of the irrigation, after two air injections to remove the water, 2 cc. of the solution was injected into the sinus and left there. This process was repeated twice more, injections being 2 days apart, and the operation done the day after the final injection.

Cat 1-R-36. Right sinus chronic infection.

The reaction in this cat was not especially acute. A moderate discharge was noted during the first week. During the second week no discharge was present, and the cat was apparently well. For 5 days during the third week a profuse sero-purulent discharge was noted, with apparent recovery the following weeks. At the end of the sixth week, the cat was anæsthetized and about 2 cc. of tetrandrine methiodide 1% solution was perfused through the sinus (until it appeared at the nostril). Three days later the cat was killed, the head cut off and the sinus membrane exposed. The specimen was then placed in Zenker-formalin for 24 hours, washed for 24 hours and then placed in 70% alcohol. At this stage, the sinus membrane was dissected from the skull. It was thickened, somewhat adherent, and contained about ½ cc. of purulent material. No gross hyperemia was noted. The membrane was then carried through the higher alcohols, xylol and embedded in paraffin. It was then sectioned at 6 micra thickness and stained according to the Maximow hemotoxylin-eosin-azur technique. Microscopically, most of this membrane was quite normal in ap-

pearance. Epithelium and cilia were normal, save for some increase in goblet cells. Tunica propria slightly thickened in part. One area considerably thickened, with accumulation of lymphocytes and plasma cells resembling old chronic membrane. Few leukocytes. Some of the blood vessels in section had many eosinophiles. Some edema present.

No. 1-L-36. Left sinus, same animal as 1-R-36.

This sinus was not infected. One perfusion of tetrandrine methiodide 1% solution was done at the same time as on the infected side and the membranes prepared in the same manner as above. In this case the membrane was not adherent. It was thin and glistening and was not hyperemic except for evidence of a slight hemorrhage at the site of injection. No exudate was present within the sinus. Microscopically, the membrane shows evidence of a mildly acute irritation with edema, fairly numerous polymorphonuclears including eosinophiles, and some lymphocytes. The epithelium is considerably injured in spots, some places almost destroyed, elsewhere the cells appear nearly normal with intact cilia. There is a mucous cyst surrounded by low epithelium. The membrane is hyperemic. The entire picture points to considerable irritation by the injection of fluid.

No. 2-R-36. Right sinus, chronic infection.

This cat was quite sick for two days following infection. A nasal discharge was present for three weeks. The cat was in excellent spirits and excellent in appearance after this time. One cc. of hordenine methiodide 1% solution was perfused through this sinus at the end of the sixth week. Dissection showed a thickened dull membrane, not as adherent as No. 1-R but containing a little more exudate somewhat thinner. Some hyperemia of the membrane was noted. Microscopically, the membrane is greatly thickened with considerable fibrosis in deep part of tunica propria. The outer region of tunica propria is filled with lymphocytes and plasma cells and some polymorphonuclears. Considerable hyperemia. The surface epithelium consists of elongated columnar cells much taller than normal with numerous goblet cells. There are many mucous cysts in various stages of development extending into the tunica propria. The cilia appear about normal in some parts of epithelium, elsewhere they are clumped together, and in some places apparently destroyed. The blood vessels of the tunica propria contain numerous eosinophiles. There are some large cyst-like structures deep in the tunica surrounded by evi-

dence of much tissue destruction. Some parts of the membrane show very great concentration of the plasma cells beneath the epithelium, but very little evidence of acute exacerbation.

No. 2-L-36. Normal sinus, same cat as 2-R.

About 1 cc. of hordenine methiodide 1% solution was perfused through this sinus. An apparently normal membrane was noted upon dissection. Slight hyperemia was present at the site of injection. Microscopically, the epithelium was normal in appearance; cilia intact; cells low columnar to cubical; very few goblet cells. Tunica propria slightly edematous in appearance and slightly hyperæmic. Injected material in sinus cavity filled with red blood corpuscles.

Cat No. 3.

This animal was prepared in the same manner as the previous two. The reaction throughout was exceptionally mild. A definite infection was obviously present, however, as indicated by a persistent watery discharge from the cat's nose. At the time of perfusion the infected right side was successfully perfused with benzyl dimethylamine methiodide 1% solution. But when the normal left side was attempted the animal stiffened, gasped, took a deep breath and ceased to breathe. In spite of prolonged and continuous artificial respiratory measures the animal would not breathe. In about ten minutes the lower extremities quivered and clonic contractions of the lower and upper extremities were noted. It was the impression of the operator at the time that he had possibly gone through the inferior or posterior wall of the sinus into the subarachnoid space or more likely that the major portion of the perfused solution had gone down the posterior nasal ostia into the lungs, the cat being fairly deep under ether anæsthesia at the time. Careful examination of the skull, however, revealed no perforation of the inner walls of the sinus. The lungs upon examination showed marked emphysema peripherally.

In the second group of cats the perfusion was similarly attempted. This time the anæsthesia was purposely made light and the head held well forward to prevent, if possible, perfusion into the posterior pharynx. An identical reaction was obtained in this animal as in the former instance. Benzyl dimethylamine methiodide 1% solution was successfully perfused through the infected sinus, but when the normal side was attempted this animal also gasped, took a deep breath and would not breathe again in spite of more prolonged artificial respiration. Clonic contractions

also set in as before. This time regurgitant vomiting occurred 5 minutes after respiration ceased. Autopsy revealed no perforation into the brain cavities, and no obstruction of the trachea or larger bronchi. Marked emphysema was noted in both lungs, especially peripherally.

The lungs were tied at the tracheal junction and fixed immediately in formalin. Microscopically, sections of the lungs show marked congestion of the capillaries and blood vessels with strong contraction of larger vessels, i.e., pulmonary artery and vein.

No other animal suffered from a reaction similar to this with any of the other solutions or in the operator's previous experience. In the third group of cats, benzyl dimethylamine methiodide 1% solution was again perfused through the sinuses of two different animals. In both these instances a response was obtained similar to that obtained using the other drugs. No unusual reaction occurred.*

No. 4-R-36. Right sinus, chronic infection.

This cat had an acute sinusitis for about 3½ weeks. It was quite sick during the first week, being lethargic and quite mean. During the second and third weeks it was more attentive and friendly, but suffered a severe head cold. It was perfused as before with beta phenylethylamine 1% solution. Upon dissection of this membrane it was noted that it was markedly thickened, dull and adherent. A large quantity of purulent material was present within the sinus. It was especially thickened and more adherent at the site of injection. Microscopically, the membrane was greatly thickened with much fibrosis, filled with plasma cells in barricade-like layer rather deep below the epithelium. Between this layer and the epithelium there is a zone containing fewer plasma cells but numerous polymorphonuclears. The epithelium is greatly thickened, has numerous goblet cells, migrant polymorphonuclears, and the cilia are partly denuded. Some mucous cysts extend into the tunica propria. The epithelium and the underlying zone give some evidence of acute irritation superimposed upon the chronic infection. Part of the membrane is extremely edematous, greatly thickened and is quite completely infiltrated with leukocytes, plasma cells, and lymphocytes. The epithelium contains very numerous migrant leukocytes many of which have

* Note by Dr. K. K. Chen, personal communication: "Beta phenylethylamine hydrochloride is slightly more toxic than ephedrine sulphate, but is decidedly much less toxic than neo-synephrine. All these figures were obtained by intravenous injection."

reached the surface. In patches the epithelium has disappeared, but there is evidence of regeneration through squamous cells.

No. 4-L-36. Same animal as No. 4-R-36 but sinus normal.

Perfused with beta phenylethylamine 1% solution. In this specimen the membrane was somewhat thickened throughout and was also somewhat dull, but at the site of injection it was much more thickened and somewhat more hyperemic than the usual normal membrane. Microscopically, the membrane was greatly thickened, slightly edematous and hyperemic. The epithelium shows destruction of its surface with only small patches of its cilia and these greatly injured. There is much proliferation of epithelium as shown by the many layers and numerous mitotic figures.

No. 11-R-36. Right sinus chronic infection.

This animal belonged to the second group. Perfused with tetrandrine methiodide 1% solution. In this case a second perfusion of the solution was done 24 hours after the first and the animal killed 72 hours after the second injection. The cat was not acutely ill at any time. A nasal discharge, however, was noted for about 1 week. After the first perfusion the cat revived well and was in apparently good health at the time of the second. The second perfusion was similarly withstood and the animal killed on the third day after it. This membrane was not markedly thickened, but somewhat adherent on dissection. Considerable hyperemia was present. Microscopically, the membrane was greatly thickened, edematous, hyperemic and filled with plasma cells. There are numerous polymorphonuclears and some eosinophiles. The epithelium has been entirely destroyed in patches, elsewhere it shows considerable injury, is tall, goblet cells not conspicuous, cilia destroyed or clumped together. There are deep crypts in which the epithelium appears more nearly normal except the goblet cells more numerous. In these crypts the cilia appear normal. Apparently no sign of repair processes.

No. 11-L-36. Left sinus not infected.

Perfused twice with tetrandrine methiodide 1% solution. In this case the membrane was not thickened but was somewhat adherent. Moderate hyperemia was present at the site of injection. Microscopically, the mucosa was thin, but hyperemic. Epithelium, low columnar to squamous-like cells without cilia anywhere; entirely destroyed in patches. Considerable number of polymorphonuclears in tunica propria, indicating a subacute inflammation.

The injury to the epithelium is the most marked feature of this membrane. Sinus cavities filled with fibrin and leukocytes.

No. 22-R-36. Right sinus, chronic infection.

Perfused twice in the above manner with hordenine methiodide 1% solution. This cat suffered an acute infection for three weeks. Afterward it subsided. The membranes were dull and thick but were freed easily from the long wall. Moderate hyperemia was present. The sinus was filled with purulent material. Microscopically, the membrane was thickened to about five times normal. Plasma cells and leukocytes scattered in tunica propria, but not very numerous. Occasional small patches of lymphocytes. Epithelium tall, pseudostratified, fairly numerous goblet cells filled with mucus, cilia intact, slight hyperemia.

No. 22-L-36. Left sinus, same cat as No. 22-R.

Perfused twice with hordenine methiodide 1% solution. Membrane thicker than a normal membrane but not adherent. Slight thickening was present at the site of injection with slight hyperemia at this point. Some slight mucous-like material was present within the sinus. Microscopically, the membrane was about normal in thickness; epithelium intact; low columnar cells with tall intact cilia; some hyperemia; and lymphocytes somewhat more numerous than normal. The cilia are destroyed in patches, and the epithelium shows signs of injury.

No. 33 CAT. Cat died as described under No. 3.

No. 44-R-36. Right sinus, chronic infection.

Two perfusions with beta phenylethylamine 1% solution. The infection in this cat was moderately acute. A discharge of purulent nature was present for two weeks following injection. This cat was sick following the first perfusion and was submitted to the second perfusion with some difficulty. For two days following this operation it was feared the cat would die, but on the third day it revived considerably and was killed in the usual manner. Dissection of the membranes was difficult because of the marked thickening and apparent friability, the membranes being very adherent to the long walls. Marked hyperemia was grossly present. Microscopically, the membrane was thickened, strongly edematous in deeper part with large spaces filled with grayish staining substance. Tunica propria somewhat hyperemic with scattered plasma cells and polymorphonuclears. Epithelium tall with numerous gob-

let cells. Cilia clumped and destroyed in patches, elsewhere fairly numerous.

No. 44-L-36. Left sinus, normal sinus, same cat as No. 44-R, two perfusions.

This membrane was more dull and thicker than the usual non-infected membrane. It was markedly thickened at the site of injection. It was also more hyperemic than usual and very similar to the membrane in which beta phenylethylamine 1% solution was used before. Microscopically, the membrane was very thin, low columnar to cubical cells; cilia clumped in many places, elsewhere no sign of injury; no hyperemia; very few cells in tunica propria.

No. 111-R-36. Chronic infection, right sinus.

One perfusion with tetrandrine methiodide 1% solution. Animal suffered a nasal discharge for one week. Membrane thickened and adherent, a moderate amount of purulent material was present within the sinus. No gross hyperemia was noted. Membrane was thickest at the site of injection.

No. 111-L-36. Chronic infection, not perfused. Same animal as No. 44-R.

Some thickening was present but not as great as the usual infected sinus. No hyperemia and very little adherence to the wall was noted.

No. 222-R-36. Right sinus, chronic infection.

Two perfusions with hordenine methiodide 1% solution. A moderately severe infection was present throughout the whole six weeks preceding perfusion. After the first perfusion the animal was quite sick for one day but revived from the second perfusion well. The membranes in this case were moderately thick and adherent; slight hyperemia was noted grossly. Microscopically, the mucosa was thickened; considerable fibrosis; plasma cells and leukocytes scattered but not numerous; deep cysts with broken-down epithelium and mucus are present; surface epithelium is tall, pseudostratified with partially destroyed cilia.

No. 222-L-36. Left sinus, chronic infection, not perfused.

Membrane quite thick and adherent. No hyperemia noted. Microscopically, the membrane was not greatly thickened, very little, if any, sign of infection; epithelium normal; cilia somewhat clumped together.

No. 333-R-36. Right sinus chronic infection, two perfusions with No. 3 solution.

Infection mild, nasal discharge for 4 days. Usual reaction when perfused. Membrane moderately adherent, and moderately dull and thick. No hyperemia was present grossly. Some hemorrhage at site of injection. Microscopically, the membrane was thin; epithelium, low columnar cells with numerous goblet cells and marked indications of injury to cilia.

No. 333-L-36. Left sinus, chronic infection, not perfused.

Membrane thickened more than normal but less than the usual chronically infected sinus. Slight thickening, slight adherence to the wall and mild hyperemia was visible throughout. Microscopically, the membrane was somewhat thickened; considerable hyperemia; epithelium tall, numerous goblet cells; cilia about normal in appearance.

No. 444-R-36. Right sinus, chronic infection, one perfusion.

The cat was quite sick the first two weeks of infection. A profuse nasal discharge persisted and the cat bordered on a pneumonia-like condition. It had apparently recovered fully at the time of perfusion at six weeks. After the perfusion the cat became sick again and remained semi-comatose until it was killed at the end of the third day. The sinus was filled with purulent exudate. It was thick and quite adherent. Hyperemia was apparent throughout. Microscopically, the membrane was somewhat thickened, rather few cells, a few extravascular eosinophiles in tunica propria; epithelium tall, numerous goblet cells. Cilia about normal. Comparatively few cells except, here and there, there are patches of polymorphonuclears. Blood vessels show numerous eosinophiles. The whole picture is one of slight injury with little change from the injection.

No. 444-L-36. Left sinus, chronic infection, not perfused, same cat as No. 444-R.

This membrane was also hyperemic, thick, dull and moderately adherent. The sinus contained a moderate amount of purulent exudate. Microscopically, typical picture of chronic infected membrane with numerous plasma cells, edema, very tall epithelium, and numerous mucus-filled goblet cells. Cilia somewhat clumped but not greatly injured.

No. 103-R-36. Right sinus, chronic infection, perfused once with benzyl dimethylamine methiodide 1% solution. This cat suffered

a mild infection with a nasal discharge for 11 days. It was in excellent health at all times. The animal was seemingly unaffected by the perfusion or anæsthetic. The membrane was moderately thickened, slightly hyperemic and quite adherent. Some hemorrhage was apparent and increased thickening was evident at the site of injection. Microscopically, typical picture of chronic infected membrane with numerous plasma cells, some edema, tall epithelium, goblet cells not conspicuous, patches of lymphocytes, epithelium destroyed in patches, elsewhere the cilia are somewhat clumped but not greatly injured.

No. 103-L-36. Left sinus, chronic infection, perfused once with tetrandrine methiodide 1% solution.

Membrane moderately thickened, slightly adherent and slightly hyperemic. Some thickening was present at the site of injection. Microscopically, greatly thickened membrane, marked edema, considerable hyperemia. Tunica propria filled with plasma cells and very numerous polymorphonuclears. Epithelium somewhat taller than normal but intact. Cilia show little sign of injury. Goblet cells more numerous than usual.

No. 2-H-36.

Human middle-aged male, history of chronic sinusitis for ten years; had been placed on chloretone and ephedrin inhalant nose drops, and later had numerous antrum irrigations over a period of about six months. Complained of headaches, frequent colds and pain over left antrum. There was radiographic evidence of marked sinus involvement. On irrigation, a considerable quantity of exudate was found in the left antrum. Two cc. of hordenine methiodide 1% solution was injected into the sinus and left there. Forty-eight hours later the process was repeated. A lesser amount of purulent exudate was present. Two cc. of solution was again injected. The patient, quizzed six hours after each injection, claimed no increase or decrease of symptoms. The process was repeated again 48 hours later. This time a very small amount of muco-purulent material was present. The patient reported no change in his condition. Twenty-four hours after the final injection the sinus membrane was removed by Caldwell-Luc operation. The membranes were somewhat adherent but the sinus wall was removed intact. It was markedly thickened and fibrous in character. No purulent exudate was present within. Microscopically, the membrane was greatly thickened, deep part is edematous

and has lymph follicles; more superficial part is infiltrated with lymphocytes and plasma cells; has every indication of an old chronic infected membrane. Lymph follicles are found beneath the epithelium, which is denuded in patches, elsewhere greatly injured showing only the basal layer of cells with the more superficial columnar cells in various stages of destruction. Occasionally small patches of columnar cells with nearly normal cilia are found. There are numerous folds of epithelium below the general surface, some forming crypt-like recesses. In these the epithelium is about normal save for some infiltration of leukocytes. Occasional plasma cells show two nuclei. There is some indication of an increased number of polymorphonuclears with some migration of polymorphonuclears through the epithelium.

No. 4-H-36.

Human middle-aged male patient. History of acute exacerbations of chronic sinusitis for at least 5 years. Patient suffered from marked pain below the left eye and a persistent left frontal headache.

As with the former patient, this man was given three antrum irrigations with sterile water, followed by injections of 2 cc. of beta phenylethylamine 1% solution. In this case, however, the patient reported marked benefit and relief of pain following the injections; in fact was on the verge of refusing operation after the third injection. As in No. 2-H, the purulent exudate was very much reduced at the time of the third irrigation. The membrane was thick, adherent and fibrous in character. Several small polyps were found within the cavity, but no purulent exudate was present. Microscopically, the membrane was greatly thickened, very edematous, vascular congestion. The membrane is infiltrated with plasma cells. Much of the epithelium is denuded, with only the basal layer of cells remaining. Here and there even the basal cells are gone, exposing the tunica propria to the surface. Elsewhere the epithelium is present as stratified columnar, but shows various degrees of destruction. Beneath the epithelium layer are found numerous cysts of various sizes.

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THE TREATMENT OF CANCER OF THE LARYNX

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BY INVITATION

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THIS REPORT is a discussion of the methods now used at the Memorial Hospital in the treatment of cancer of the larynx. These consist of surgery and radiation, either alone or in combination. In our choice of the treatment method for an individual case of laryngeal cancer, we are not concerned with any attempt to prove the superiority of either radiation or surgery. In our opinion, these methods need not be considered in a competitive sense, since each is clearly indicated in entirely different anatomic forms of the disease. The Clinical Staff at Memorial Hospital is composed largely of surgeons who have entire charge of the treatment, whether it be by surgery, radium or x-rays, and the surgeon-radiologist is therefore able to use his unbiased judgment in the selection of the particular method or combination of methods best suited to the individual cancer patient.

Classification. Anatomically, cancer of the larynx is divided into two general varieties: (1) intrinsic, (2) extrinsic. (There are sometimes added (3) mixed intrinsic and extrinsic, and (4) subglottic). An anatomic classification of cancer of the larynx is of great value in predicting the clinical course, and in selecting the method of treatment in the individual patient. There is some difference of opinion as to the exact anatomic landmark separating cancers of the intrinsic and extrinsic larynx. Many writers are rather vague on this point, and include as intrinsic cancer the lesions arising on the posterior surface of the epiglottis and inner surfaces of the aryepiglottic folds. St. Clair Thomson[1] considers as cancers of the intrinsic larynx all growths arising on the vocal cords, the ventricles and the ventricular folds. He classifies as cancers of the extrinsic larynx all growths arising on the arytenoids, the post-cricoid region, aryepiglottic folds and epiglottis.

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We believe that the line of demarcation between cancers of the intrinsic and extrinsic larynx should be the free borders of the ventricular folds, so that the term "cancer of the intrinsic larynx" would be limited to the tumors arising on the vocal cords or in the ventricles, and all the lesions arising on the ventricular folds or above should therefore be classified as extrinsic growths (Fig. 1). The fact that cancer almost never arises on the free edge of the ventricular fold is an additional reason for using it as the line of demarcation.

In our opinion, the anatomic classification should be based on the point of origin of the growth; that is, if the lesion begins on a vocal cord, it should subsequently always be classified as cancer of the intrinsic larynx, even though it may later extend upward

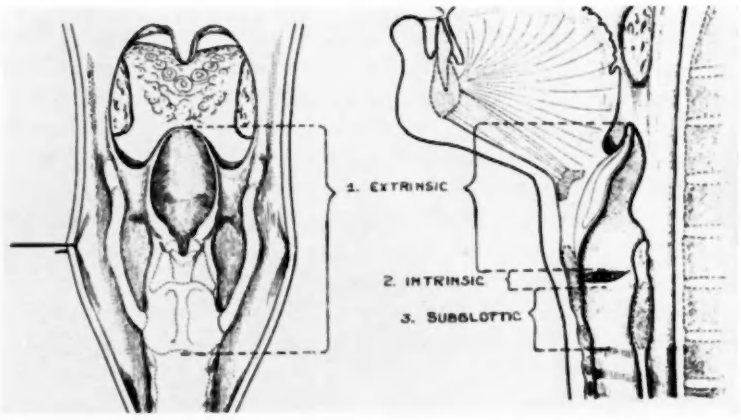

FIG. 1 — (Martin) — Anatomic classification of laryngeal cancer.

to invade the structures of the extrinsic larynx, or perforate the cartilage to invade the subcutaneous tissues. For example, in correct medical usage, one refers to cancer of the tonsil as such, even after the growth has extended onto the pharyngeal walls, the soft palate and to the cervical nodes. By such terminology, we are able to designate the essential nature of the disease process, and from past experience in cases of similar origin, to predict the probable clinical course. For the same reason, we may advantageously go a little further and classify tumors as to their points of origin within one anatomic structure, and therefore two growths arising in the intrinsic and extrinsic larynx, respectively, would each retain its original designation, though both might later extend

widely throughout the larynx. If one accepts such a basis for classifying cancer of the larynx, the term "mixed intrinsic and extrinsic cancer" is superfluous and confusing. Personally, we do not use the term.

Subglottic laryngeal cancer is usually considered as a subgroup of the intrinsic variety. These tumors are at times difficult to differentiate from growths arising on the tracheal walls and extending upward to invade the vocal cords.

Tumors arising on the vocal cords differ markedly from those of the extrinsic portions of the larynx in their symptomatology, clinical course, histopathology, radiosensitivity and curability by various methods of treatment. As a group, the growths arising in the vocal cords, the ventricles or in the subglottic region have about the same symptomatology, clinical course, histopathology and radiosensitivity, and require about the same methods of treatment. Those arising on the ventricular folds or above form another group, in which the above-mentioned characteristics differ markedly from the intrinsic group. We shall later discuss these characteristics at greater length.

Incidence. It is usually stated that the intrinsic larynx is the site of origin of 65-70 per cent of all laryngeal cancers. In our clinic, the reverse is true, and the proportion is intrinsic 25 per cent and extrinsic 75 per cent.

It has been suggested that one who confines himself largely to the practice of laryngology will necessarily find a great preponderance of his cases to be of the intrinsic variety, since hoarseness and dyspnœa are early symptoms, which will naturally cause the patient to be referred to a laryngologist. Growths of the extrinsic larynx produce hoarseness and dyspnœa late in the course of the disease. Cervical adenopathy and dysphagia are common early symptoms, so that these patients are more apt to be referred to the general surgeon, and may go on to a fatal termination under a diagnosis of cancer of the hypopharynx or pharyngeal wall. Our Head and Neck Service is made up of all patients with cancer above the level of the clavicle who apply to Memorial Hospital. The final diagnoses are made only after careful clinical and pathological study. It is also possible that our high percentage of extrinsic cancer is due in part to the fact that early intrinsic growths of the larynx are frequently operated upon by laryngeal surgeons in general hospitals, and that cancer of the extrinsic larynx, being inoperable, is more often referred to a cancer hos-

pital, such as ours. In any case, we believe the incidence of extrinsic cancer of the larynx to be far greater than heretofore recognized.

Histologic diagnosis. A histologic confirmation of the diagnosis is essential before subjecting the patient to the necessarily aggressive treatment for laryngeal cancer, whether by surgery or irradiation. In our opinion, the dangers attendant upon the careful removal of a small biopsy are mainly theoretical and often exaggerated. Biopsies from the intrinsic larynx or subglottic region require direct laryngoscopy. From the extrinsic portions of the larynx, we have found that tissue can be obtained by the use of a laryngeal mirror and a curved biopsy forceps more easily and with far less discomfort than by direct laryngoscopy. By the mirror, one is able to view the hypopharynx in its normal relaxed position by binocular vision. Examination through the direct laryngoscope distorts the normal relationship of the parts and permits only monocular vision through a narrow aperture.

CLINICAL, ANATOMICAL AND PATHOLOGICAL FINDINGS

Cancer of the intrinsic larynx. Practically all growths of the intrinsic larynx arise on the free edge and upper surface of the vocal cords in their anterior two-thirds. The point of origin is almost always found to be well anterior to the vocal tubercles. The disease seldom arises exactly in the anterior commissure, and we have never seen a case in which the growth began posterior to the vocal tubercle or in the interarytenoid space. Occasionally, the point of origin seems to be in the ventricle itself. As the growth progresses, it tends to extend forward and across the anterior commissure to involve the opposite cord, and upward into the ventricle to invade the ventricular fold secondarily. The disease may perforate the cartilage quite early or extend through the thyrohyoid or cricothyroid membranes and produce a fluctuating infected abscess invaded by cancer in the subcutaneous tissues in this region. Hoarseness is invariably the first symptom of cordal cancer, and next follows dyspnœa, which may be a comparatively late symptom. On the whole, the disease tends to progress slowly, and in the cases referred to us, we have found that an average period of about ten months elapses between the first symptom (hoarseness), and their admission to the clinic for confirmation of the diagnosis. Metastases rarely occur, and the dis-

ease is either cured or it progresses until death is caused by local extension.

Histologically, cancer of the intrinsic larynx is almost always squamous carcinoma, grades I or II. Comparative radioresistance is to be expected in these intrinsic growths. Papilloma may occur, but is comparatively rare in the adult. The diseases offering the greatest difficulty in differential diagnosis are, in our experience, in the order of their frequency, tuberculosis, pachydermia, benign polyp, papilloma and syphilis.

Subglottic cancer resembles the intrinsic variety histologically. Clinically, the diagnosis is apt to be made rather late, since the tumor may reach a larger size before producing marked symptoms. Mirror examination of the larynx may fail to reveal the growth, and the symptoms may be confused with asthma or cardiac dyspnœa. Spasm of the larynx is a very frequent symptom, and we have learned to suspect a subglottic growth when laryngeal spasm, accompanied by severe paroxysms of coughing and cyanosis is brought on by laryngeal examination. In these cases, although cancer may be suspected, an exact diagnosis may be impossible without laryngotomy.

Cancer of the extrinsic larynx. Under this classification, we include all growths arising on the upper surfaces of the ventricular folds, the epiglottis, the aryepiglottic folds, the arytenoids, the post-cricoid space, and the laryngeal surfaces of the pyriform sinuses. At times, it is difficult to determine the exact point of origin of widespread extrinsic lesions, and it may only be possible to make an accurate anatomic diagnosis as the lesion is seen to regress under radiation therapy. These growths differ markedly from those of the intrinsic larynx in their symptomatology, clinical course and usual histopathology.

Hoarseness and dyspnœa never occur as early symptoms. If the lesion arises on an arytenoid or ventricular fold, it is apt to cause dyspnœa before dysphonia. If the tumor arises on the arytenoids, the aryepiglottic folds or the epiglottis, it may reach a size of 3-4 cm. in diameter without producing any marked local symptoms. Ordinarily, the first symptom complained of is a sense of a "lump" in the throat, and some difficulty in swallowing. If there is ulceration, slight hemoptysis may occur fairly early in the course of the disease.

The clinical course is more rapid in the extrinsic than in the intrinsic form of laryngeal cancer. Metastases tend to develop

quite early, usually first in the upper deep cervical nodes (carotid bulb), later extending to the lower deep cervical nodes or diffusely throughout the neck. Even if the disease is controlled locally, systemic metastases (lungs, bone, etc.) are prone to develop after periods of 1-5 years or even longer. This tendency toward early regional and late generalized metastases is one of the most important factors in the relatively poor prognosis in extrinsic laryngeal cancer. It is not uncommon for the development of cervical nodes to be the first symptom noted by the patient in cancer of the extrinsic larynx. We have repeatedly seen patients with nodes over the carotid bulb 3-4 cm. in diameter in whom the primary growth in the extrinsic larynx, found on a routine examination, produced no symptoms whatsoever. At other times, the primary lesion may remain quite small, and death may occur from the effects of the metastases alone.

Histologically, cancer of the extrinsic larynx includes a large percentage of such anaplastic varieties as lymphoepithelioma, transitional cell carcinoma or anaplastic epidermoid carcinoma, grades II or III. In our experience, such tumors are never found to arise on the cords themselves. Adult squamous carcinoma is not uncommon in the extrinsic larynx, but epithelial pearls are rarely found. Comparative radiosensitivity is characteristic of the growths of the extrinsic larynx in contradistinction to the radioresistance of intrinsic growths.

SELECTION OF THE METHOD OF TREATMENT

In the treatment of laryngeal cancer, we have at our disposal surgery, radium and x-rays. In our opinion, no single one of these alone is suited to the treatment of all the clinical forms and histologic varieties of this disease. In some instances, surgery alone is the method of choice, while in others, radiation alone offers the best or the only chance of cure. There are still other situations in which only a combination of surgery and radiation is adequate to control the disease.

For the sake of clarity, we have outlined our several techniques in Table I. One cannot categorically recommend any one single procedure for any one clinical or anatomic variety of laryngeal cancer. Some lesions do not fit exactly into a definite clinical classification, and may therefore require considerable variation in technique and clinical management.

TABLE I

TREATMENT METHODS OF LARYNGEAL CANCER

(1) *Partial laryngectomy* for intrinsic cancer—early or moderately far advanced.

(2) *Laryngostomy and implantation of radon seeds* for advanced cancer of the intrinsic larynx, especially those with perforation through cartilage, the thyrohyoid or cricothyroid membranes.

(3) *Protracted endolaryngeal radiation with the laryngostat* for certain intrinsic growths.

(4) *External radiation by the divided or fractional dose principle* for all extrinsic laryngeal cancer. Supplementary interstitial radon may be used for metastatic nodes.

I. PARTIAL LARYNGECTOMY (LARYNGOFISSURE, LARYNGOTOMY, THYROFISSURE, THYROTOMY, HEMILARYNGECTOMY)

Definition. To define an operative procedure which consists of the removal of a portion of the intrinsic larynx of either one or both sides, we use the term "partial laryngectomy," an antonym of total laryngectomy. The term is not entirely satisfactory in that it does not specify whether only one cord is removed or whether parts of both cords and a segment of the cartilage are also excised, but we have been unable to settle upon a better one.

The classic procedure which consists of opening into the laryngeal cavity by splitting the thyroid cartilage in the midline and subsequently removing a portion of the structures of the intrinsic larynx, with or without a partial resection of the thyroid cartilage, has been variously named laryngofissure, laryngotomy, thyrofissure and thyrotomy. Etymologically considered, these terms can refer only to the preliminary step of opening the larynx, and are no more specific than if "laparotomy" were used to signify what we commonly designate as "appendectomy" or "cholecystectomy." The more important and significant part of the procedure, that is, the excision which is carried out after the interior of the larynx is exposed, is not expressed by any of the above-mentioned terms, all of which could be just as fairly used if the larynx were simply opened, inspected and closed. Many authors express their preference for "partial laryngectomy," but nevertheless continue to employ the term "laryngofissure."

Hemilaryngectomy is sometimes used to define the operation of partial removal of the soft tissues of one side of the intrinsic larynx, including a major portion of one thyroid cartilage. In our

opinion, this term may not be logically used unless half of the epiglottis, half of the cricoid and one arytenoid are also removed. The latter procedure has been advocated, but we can see no clear indication for its use from the standpoint of the anatomic forms of cancer of the larynx. "Anterior laryngectomy" is used by Patterson[2] to define the removal of the anterior portion of both cords and cartilages for a lesion at the anterior commissure. Partial hemilaryngectomy, a rather cumbersome term, might be used to indicate a partial operative excision limited to one side.

Indications for partial laryngectomy. This procedure is, we believe, the method of choice in the treatment of early or moderately advanced cancer of the intrinsic larynx. At this point, we shall digress briefly to consider the possibilities and limitations of other methods in early intrinsic cancer, namely, radium implantation and protracted x-radiation, which later in this report are recommended for other clinical and anatomic forms of the disease. The question also arises as to the value of and indications for total laryngectomy as opposed to partial laryngectomy for operable intrinsic cancer.

Radium implantation, either through the laryngoscope or after laryngotomy, is not suitable to early or moderately advanced cancer of the intrinsic larynx, in our experience, although it may occasionally be successful. Implantation through the laryngoscope is inexact, since monocular vision is insufficient to permit of the three dimensional conception of the tumor so essential in exact interstitial implantation. Cancer of the intrinsic larynx is highly differentiated histologically and comparatively radioresistant, and for its control by radiation, the local intensity must be such as to produce marked changes in the local structures, namely, the underlying cartilage and the soft tissues at the level of the glottis. Radionecrosis is particularly apt to occur in the intrinsic larynx, since the blood supply from the base is interrupted by the thyroid cartilage. The circulation is therefore entirely peripheral — always a significant factor in radionecrosis. For these reasons, slight radionecrosis and perichondritis are prone to develop in the intrinsic larynx following interstitial implantation. Such complications might not be particularly serious in other portions of the larynx, but at the glottis itself, they are of serious import without laryngostomy to provide drainage and to relieve obstruction. For these reasons, we advise against the attempts at cure of early intrinsic cancer by implantation of seeds through the

laryngoscope. We reserve radon implantation in laryngeal cancer to those cases where the disease has extended widely, perforated cartilage, or invaded the subcutaneous tissues. In such cases, we have no choice, since there seems to be no other method, and we accept the above-mentioned complications and long morbidity as inevitable.

When Coutard first demonstrated the results of protracted x-radiation in the treatment of cancer of the pharynx and extrinsic larynx, it was not generally appreciated that the success of this principle was dependent upon the comparatively high radiosensitivity of certain growths which are never found in the intrinsic larynx itself. Cordal cancer is highly radioresistant and can seldom be made to regress completely under protracted x-radiation alone. Furthermore, the immediate or late complications in the intrinsic larynx (radionecrosis, etc.) which commonly follow the large total doses of protracted x-radiation necessary for the control of these radioresistant lesions almost always terminate fatally. Once radionecrosis has developed in the intrinsic larynx following the average protracted x-radiation dosage, we have never been able to prevent a diffuse spread of the slough and eventual death. These complications are much less frequent and of far less significance in the extrinsic larynx or pharynx.

Limitations and disadvantages of total laryngectomy. We do not employ total laryngectomy in the treatment of laryngeal cancer. In our opinion, this radical procedure may be dispensed with if partial laryngectomy is intelligently modified to care for all early and moderately advanced lesions, and laryngostomy and radon seed implantation is employed for the lesions too far advanced for partial laryngectomy. From the standpoint of cure alone, we do not question the success of total laryngectomy as it has been perfected by MacKenty and Buckley in intrinsic cancer confined to the laryngeal box. We do question, however, the necessity for such a radical operation in early or moderately advanced cases, and believe that the operable borderline cases are relatively few in number. As regards pharyngolaryngectomy, we are in entire accord with St. Clair Thomson[1] that "it will generally be considered that such an operation is not justifiable."

In our opinion, partial laryngectomy is equally successful in the cure of early or moderately advanced lesions, and if the disease is so advanced as to extend into the extrinsic larynx, it has also probably perforated the cricothyroid or thyrohyoid mem-

branes into the subcutaneous tissues, and has therefore become incurable by total laryngectomy as well.

As between partial and total laryngectomy, there is a wide difference in mortality, morbidity and the degree of maintenance of normal function. Total laryngectomy is an operation of considerable magnitude with a mortality and morbidity far greater than in partial laryngectomy. Furthermore, total laryngectomy carries with it an inevitable complete and permanent loss of phonation, except by the development of an esophageal voice or the use of a mechanical artificial larynx. Although the more intelligent and perseverant patient commonly adapts himself to one or the other of these expedients, we have found that the average clinic patient, even the more intelligent, often finds himself psychologically unable to employ either of these, and this handicap to normal social intercourse commonly results in marked mental depression and economic dependency. We do not question the fact that such functional defects may be justified in order to save the life from a lethal disease. The question is whether the same end may not be obtained by less radical procedures with at least partial maintenance of normal function.

From the psychological standpoint, it is both curious and significant to observe how desperately the partially laryngectomized patient struggles to retain whatever remnant of normal function he may have by forcing air into the pharynx through the narrowest sinus from the trachea or larynx so as to produce an audible whisper or hoarse toneless phonation. If due to temporary swelling or edema, this sinus tract is obstructed, the average patient becomes more concerned about his loss of speech than about the possibility of recurrence of his disease. Beck and Guttman have related[3] how one of their patients whose sinus tract had closed re-established it himself by a heated icepick. In the event of spontaneous closure of the laryngopharyngeal sinus, they advise a similar use of the actual cautery by the surgeon. In our experience, we have never seen a patient with any communication between the larynx and pharynx who evinced the slightest interest in either the development of an esophageal voice or in a mechanical larynx.

It is inevitable, therefore, that there should arise a conservative school of thought as to the necessary extent of the removal of tissue in intrinsic laryngeal cancer (Patterson,[2] Houtant and Ombrédanne,[4] Charschak[5] and others). The disease is essentially a local process whose limits may be accurately determined within 3-5 mm., provided that it has not perforated the cartilage or the

cricothyroid or thyrohyoid membranes. There is therefore nothing to be gained from a removal of more than the growth itself and a margin of 5-7 mm. of apparently normal tissue. The thyroid cartilage underlies practically the whole intrinsic larynx, forms a natural barrier to the deep extension of the disease, and furnishes an exact line of cleavage for safe and adequate excision of the base of the tumor. The cartilage may be totally removed without any significant additional operative risk. The advisability and disadvantages of cartilage removal are considered in greater detail under a description of the technique.

Partial laryngectomy may therefore be extended so as to remove both cords and both ventricular bands, the major portion of both cartilages and the cricothyroid membrane. So long as some of the mucous membrane on the posterior wall of the larynx remains, there will persist in the healed state a communicating tract between the laryngeal cavity and the pharynx, permitting of some degree of whispered or spoken voice. After wide excision of one cord and one cartilage (usually termed laryngofissure), the mortality should be very small and the morbidity short. The patient is usually out of bed on the second day after operation. He may be discharged from the hospital about the seventh to the tenth day and often returns to his regular occupation after three to four weeks with a whispered voice. Phonation returns in about six to eight weeks.

If the extent of the growth permits of such a conservative operation, we know of no procedure in a serious form of cancer which is more satisfactory or has a higher percentage of success than this one. Unfortunately, this simple standard procedure without modifications and attended by so few disadvantages is applicable only to lesions of the anterior two-thirds of one cord. If the disease has crossed the anterior commissure or extended widely up or down, certain modifications are required which do not permit of ease of performance and the degree of functional maintenance of the simpler procedure. If the disease has crossed the anterior commissure to involve the opposite cord, or if it has arisen directly in the commissure to involve both cords to about the same extent, the operation may be extended to excise widely all of the involved tissues. If the area of excision includes a portion of both cords, phonation is usually permanently lost, although the air space at the glottis may be entirely adequate so that no tracheotomy tube is required. One whole wing of the cartilage may be removed and one-half of the remaining wing excised

without decreasing the lumen at the level of the glottis to such an extent that a permanent tracheotomy is required for supplementary purposes in the average case.

TECHNIQUE OF PARTIAL LARYNGECTOMY

The details of the operative procedure are, we trust, adequately described in our illustrations and their attached legends (Figs. 2, 3, 4 and 5). The technique we use is based on the classical procedure described by St. Clair Thomson.[1] We have included certain modifications devised by others as well as ourselves. Certain factors in the technique are somewhat controversial or are modifications, and therefore require special consideration.

Anaesthesia. No preliminary medication (morphine, etc.) should be given. Prompt post-operative recovery of consciousness and of the cough reflex are of the greatest importance in avoiding pulmonary complications.

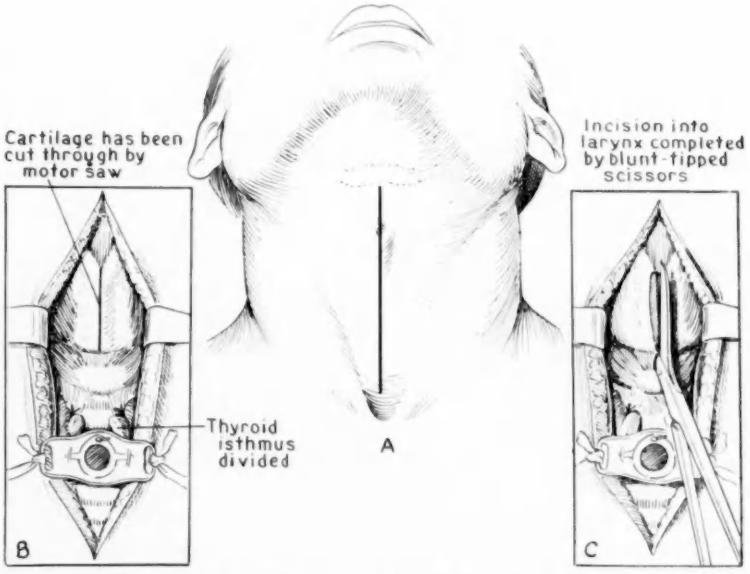

FIG. 2 — (Martin) — Partial laryngectomy. (A) An incision is made in the midline extending from the hyoid bone to the suprasternal notch. (B) The soft tissues are divided in the midline, exposing the thyroid and cricoid cartilages and the upper portion of the trachea. The isthmus of the thyroid gland is divided between ligatures. A tracheotomy tube is inserted. The thyroid cartilage is divided in the midline with a motor saw, care being taken that the laryngeal cavity is not entered. (C) The laryngeal cavity is opened by the scissors, the lower blunt-tipped blade of which is inserted through a stab wound in the cricothyroid membrane, care being taken that the cut is made directly in the anterior commissure.

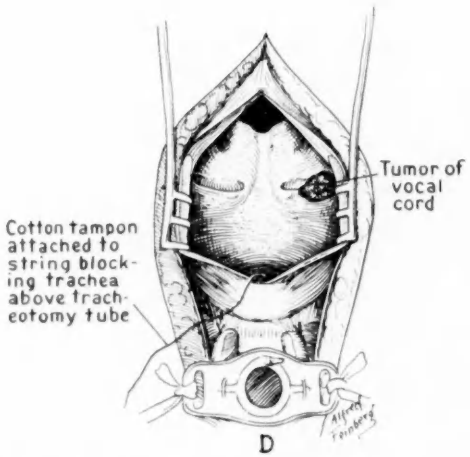

FIG. 3 — (Martin) — Partial laryngectomy. (D) After the exposure has been completed, the edges of the wound are held apart by a self-retaining retractor and a cotton tampon attached to a string is inserted into the trachea above the tracheotomy tube so as to prevent blood from entering the trachea and bronchi.

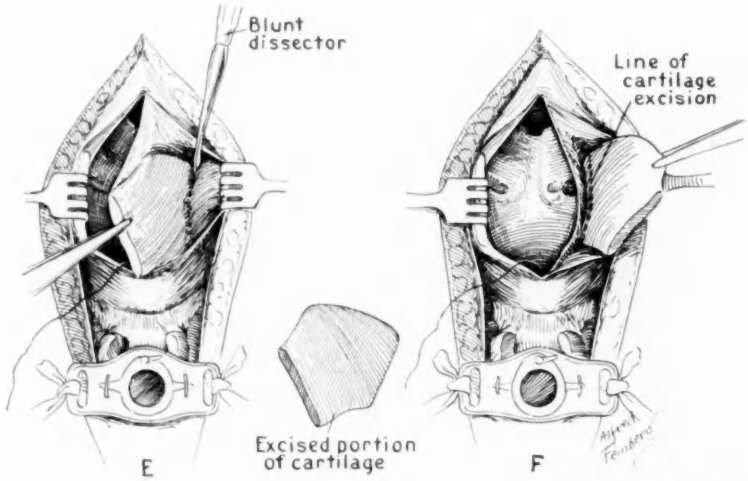

FIG. 4 — (Martin) — Partial laryngectomy. (E) Before excising the tumor and soft parts, the major portion of one thyroid ala is removed. By blunt sub-perichondrial dissection, the outer surface of the cartilage is exposed to within about one centimeter of its posterior border. (F) The inner surface is similarly exposed. The major portion of the cartilage is then excised with a scissors.

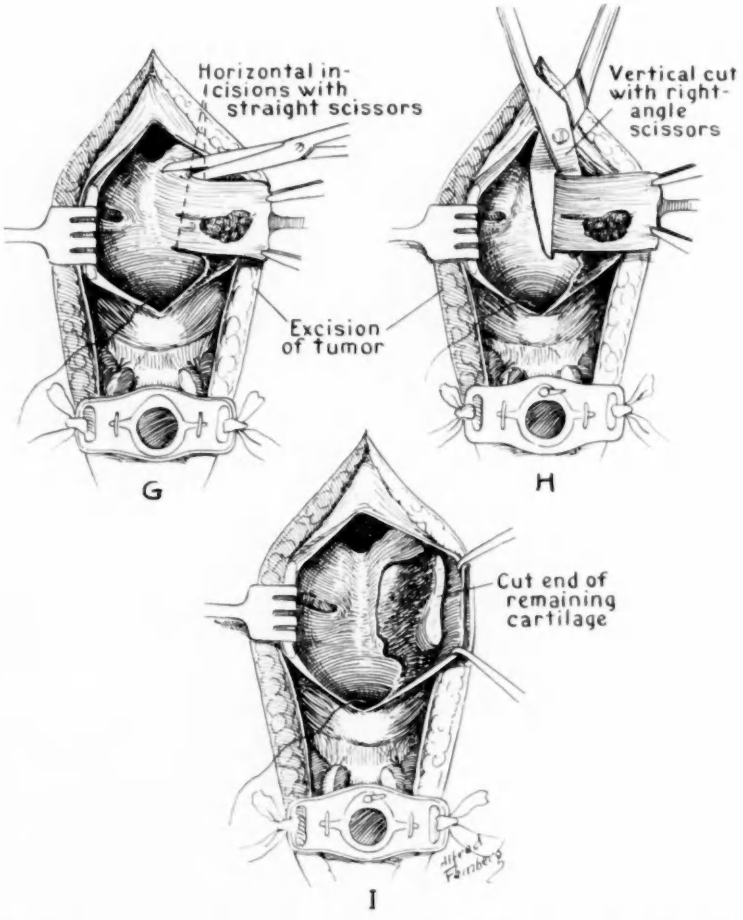

FIG. 5 — (Martin) — Partial laryngectomy. (G) The soft tissues and the tumor are excised by two horizontal incisions with a straight scissors extending back to well beyond the posterior limit of the tumor. (H) The excision of the block of soft tissue is completed by a vertical cut with a right-angle scissors. (I) Appearance of the operative wound after the excision has been completed.

We have performed the operation under local anæsthesia, but find that the cough and swallowing reflexes are impossible of absolute control by a local anæsthetic, and we believe that absolute stillness with absence of coughing or swallowing is essential in order that an accurate and adequate excision of the growth can be made. As has been recommended by St. Clair Thomson, we prefer chloroform to ether for the following reasons. There is less bronchial irritation, and the degree of anæsthesia

is more easily controlled, in that with chloroform the patient may be kept quite lightly anæsthetized throughout the operation and quickly pushed to a deeper state of narcosis with a breath or two of a more concentrated vapor if he becomes too light. Properly given, the degree of anæsthesia is so adjusted that the patient regains consciousness and the ability to cough and expel any blood clot from the trachea or bronchial tree as the last skin sutures are completed.

The patient is first anæsthetized by a mask or cone, and after draping, the anæsthetic is continued through a nasal tube inserted as far as the pharynx. Later, during the course of the operation, after the tracheotomy tube has been inserted, the anæsthetic vapor is blown gently into the tracheotomy tube through a loosely fitting sterile rubber tube.

The distrust with which chloroform anæsthesia is commonly regarded in American surgical practice may be due to the fact that we are more accustomed to ether. Chloroform is dangerous if administered with the same technique as ether. In our clinic, we are forced to employ the non-inflammable chloroform rather than ether in those cases where the actual cautery is used about the oral cavity. We have found it quite safe and satisfactory, provided that the open cone or mask is used, or that the anæsthetic vapor is produced by bubbling oxygen rather than nitrous oxide or carbon dioxide through the liquid chloroform. Rebreathing, as in ether anæsthesia, or even slight cyanosis is risky in chloroform anæsthesia, and since the safety zone is narrower than with ether and the action of the anæsthetic is more rapid, the patient should be kept on the light rather than the deep side of the safety zone.

Preliminary tracheotomy. After the superficial exposure of the larynx and trachea and before opening the larynx, we always insert a tracheotomy tube three or four tracheal rings below the cricoid. The anæsthetic is then continued through the tracheotomy. This we believe to be an important step in the procedure. With a general anæsthetic, it dispenses with the necessity for any anæsthetic tube in the operative field in the larynx itself, and also permits of plugging the trachea above the tracheotomy tube with a cotton or gauze tampon so that no blood or secretion can enter the trachea from the operative field. The tracheotomy tube is left in place when the wound is closed. When the patient has been returned to bed, a small caliber sterile catheter may be inserted through it

to protrude 1 or 2 cm. into the trachea, and constant suction may be maintained for the first few hours so as to remove the slight though inevitable ooze from the operative field down into the trachea. In case of post-operative hemorrhage, the presence of the tracheotomy tube permits of opening the incision and packing the larynx. We ordinarily remove the tube at about midnight of the day of operation. Once the patient is fully recovered from the anæsthetic and the local shock to the tissues, he is better able to expel the tracheal and bronchial secretions through the normal channels without the tube in place.

Some surgeons, Jackson,[6] Tucker[7] and others, do not employ preliminary tracheotomy as a routine. They employ local anæsthesia and do not usually remove any of the cartilage. Under these conditions, one might profitably dispense with a preliminary tracheotomy, but with a more extended procedure under general anæsthesia, we believe it to be almost indispensable and without any serious disadvantage.

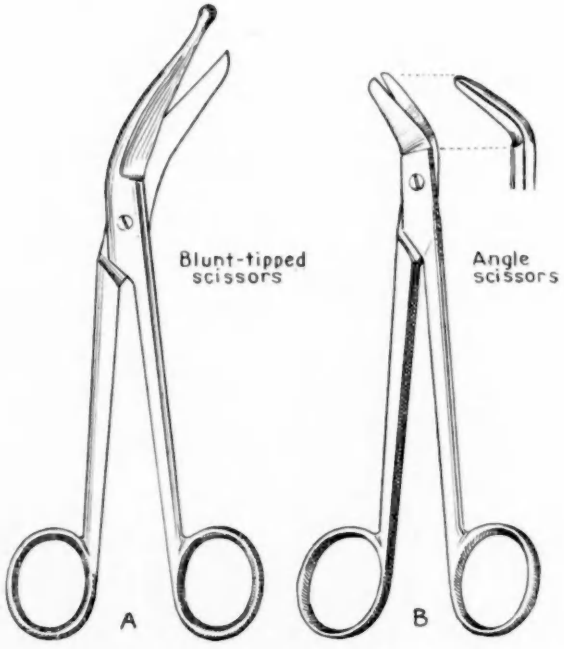

FIG. 6 — (Martin) — Special instruments employed in partial laryngectomy. After division of the cartilage by a motor saw, the blunt-tipped scissors (A) is used to complete the division of the soft tissues exactly in the anterior commissure. The beaded end of the lower blade of the scissors prevents laceration of the cord or of the tumor as the instrument is passed through the glottis from below. The angle scissors (B) are used for the vertical cut in excision of the tumor and soft parts.

Dividing the cartilage (Fig. 2). Guttman[8] has applied the motor saw to divide the cartilage in the midline. We have found this procedure to be far superior to the hand saw. The cut is quickly made without trauma to the internal structure of the larynx, which may then be divided in the midline accurately with the scissors.

Opening the larynx. In opening the larynx, one blade of a scissors or turbinotome is inserted through a stab wound in the cricothyroid membrane (Fig. 2). We have occasionally had some difficulty in avoiding laceration of the soft tissues by the sharp point of the instrument, especially if the growth has caused marked distortion within the laryngeal box. To overcome this difficulty, we have devised the instrument illustrated in Fig. 6. The beaded end of the lower blade will insure its atraumatic passage through the glottis from below upward so that the cut may be made exactly in the anterior commissure. After the larynx has been opened, we keep the laryngeal cavity and the operative field clear of blood by the use of a rubber-tipped suction tube attached either to a suction pump or to a water syphon. We find this method more satisfactory than the use of gauze sponges, since it may be used practically continuously until the larynx is closed without interrupting or seriously interfering with the operative procedure.

Removal of cartilage (Fig. 4). As we have previously mentioned, the presence of the thyroid cartilage underlying the vocal cord and the ventricle forms a natural and fairly efficient barrier to deep extension of the disease. From the operative standpoint, the mesial surface of the cartilage provides a plane of cleavage along which the soft tissues of the intrinsic larynx may be isolated in the vertical plane rapidly and almost bloodlessly by blunt subperichondrial dissection. The mesial surface of the cartilage is therefore always exposed in partial laryngectomy.

There exists some difference of opinion as to whether the portion of the cartilage thus exposed should be excised preliminary to excision of the tumor and adjacent soft tissues. The portion of cartilage to be removed consists of an entire ala except for a narrow strip of the posterior border and the cornua. Tucker,[7] Jackson,[6] Fraser,[9] Patterson,[3] New[10] and others do not always remove the cartilage for the reason that they believe that its presence helps in maintaining the normal relations and functions of the larynx. We disagree with the practice of leaving the partly denuded cartilage in place and question the validity of the argu-

ment advanced for its retention. We believe that the partial removal of the cartilage is highly desirable for several reasons. *First,* its removal permits of better exposure and facilitates the excision of the posteriorly placed soft tissues of the intrinsic larynx. *Second,* the presence of an exposed area of thyroid cartilage (calcified in most aged subjects) necessarily denuded of its perichondrium will delay healing of the operative site not only because the exposed surface is avascular, but because the cartilage prevents contracture of the wound edges — a natural and significant factor in the healing of open wounds. *Third,* the presence of both alæ of the cartilage is not necessary to maintain the patency of the glottis. After removal of one wing of the thyroid cartilage, the opening at the glottis is *larger than normal.* If the cartilage is left in, the opening is even larger still. As a matter of fact, it may be too large, so that the cord of the remaining side of the larynx cannot approach close enough to the opposite side for phonation. *Fourth,* the denuded cartilage may sequestrate and be extruded, thereby needlessly prolonging the morbidity. As long as there is any unhealed surface in the larynx not covered by epithelium, granuloma may develop. This complication, though not serious, is more common with exposed cartilage. *Fifth,* if the growth is situated in or quite near the anterior commissure, it must be realized that the soft tissues overlying the cartilage in this region form a very thin layer, so that the cartilage is very apt to be invaded, and its removal is a safeguard against recurrences from this cause.

If the major portion of the cartilage on the side of the greatest involvement has been removed, one should attempt to leave at least half or two-thirds of the remaining cartilage (Fig. 7); otherwise, the healed air space at the glottis may be insufficient without a tracheotomy. However, in the attempt to retain function, no concession should be made to adequate excision of the disease.

Excision of the tumor and surrounding soft tissues. After the two horizontal cuts with the straight scissors, as illustrated in Fig. 5, the segment of tissue containing the growth is completely removed by a vertical cut with an angle scissors (Fig. 5). The soft tissues on the inner surfaces of the thyroid cartilage are wedge-shaped in horizontal section. The apex of the wedge is at the anterior commissure and the base posteriorly at the arytenoid cartilage. We have found it of advantage to make a preliminary vertical incision partially through the vocalis muscle with the edge

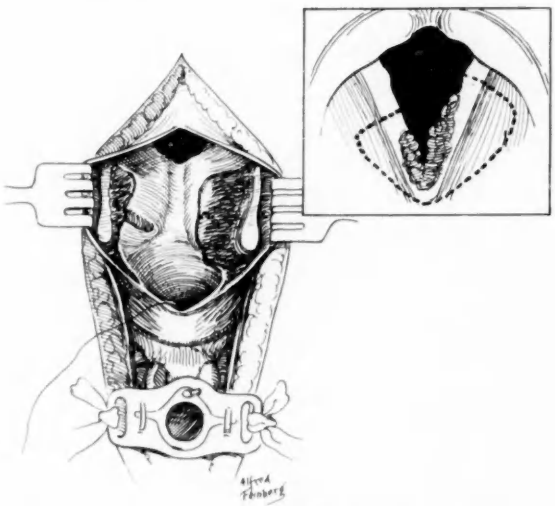

FIG. 7 — (Martin) — Partial laryngectomy. (Variation in procedure) When the growth has extended past the anterior commissure to involve both cords, partial laryngectomy may still be done, and depending on the extent of the excision, a permanent tracheotomy may or may not be required. In any case, there will always remain an air passage connecting the trachea with the pharynx, permitting of some degree of spoken voice.

of the scalpel turned toward the arytenoid cartilage so as to reduce the thickness of the tissues at this point before completing the excision with the angle scissors.

If the growth extends posterior to the vocal tubercle, the cutting edge of the scissors should be depressed posteriorly so as to cut through and remove the vocal process of the arytenoid cartilage. The question as to the posterior or upper limit of the excision or the removal of part of the cricothyroid membrane in more widespread involvement by the growth is a matter of fine judgment and experience of the surgeon.

The management of the anterior commissure when the disease invades it or approaches it closely is an important factor in the success of partial laryngectomy. If the growth definitely extends onto the opposite cord, the area of excision must obviously be extended so as to include it. In other cases, where involvement past the commissure is not quite so definite, one is sometimes reluctant to produce unnecessary deformity and additional lack of function. In these doubtful cases, we have used one of two procedures. We may insert one radon gold seed of about 1.50 millicuries, or we may sear this questionable area at the commissure with the fine point of an actual cautery (Fig. 8).

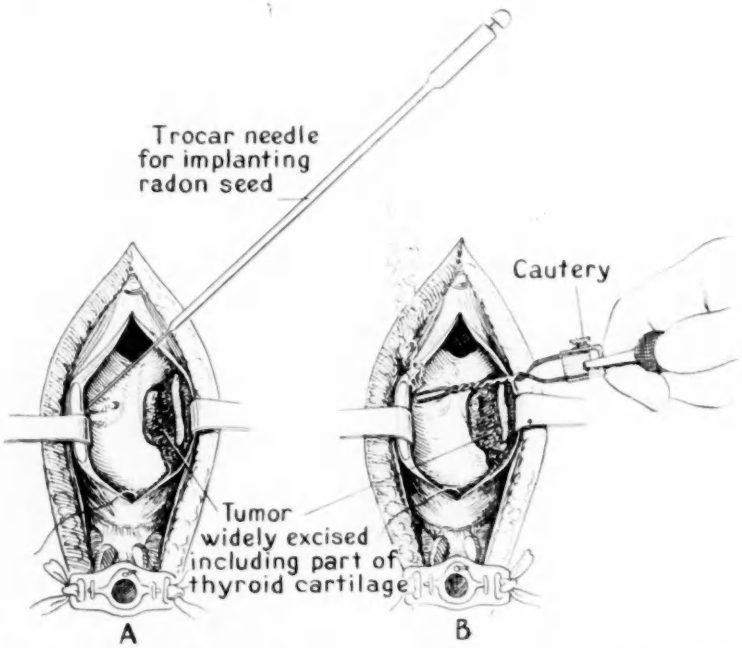

FIG. 8 — (Martin) — Partial laryngectomy (Variation in procedure). If the disease has extended to but not definitely beyond the anterior commissure, this doubtful area may be treated in one of two manners — (A) A radon seed may be implanted or (B) the actual cautery may be used to sear the tissues at this point.

Closure of the wound. No attempt is made to close the opertive defect within the larynx. The external wound is closed in two layers — the soft tissues by interrupted No. 0 chromic catgut, and the skin and subcutaneous tissues by interrupted dermal sutures. The tracheotomy tube is left in the lower angle of the wound to be removed in 12-24 hours. In suturing the soft tissues, an attempt should be made to approximate the external perichondrium of the cartilage which remains to the external perichondrium of the side from which the cartilage has been removed. If the anæsthesia has been properly administered, the patient recovers consciousness and the ability to cough as the last skin sutures are being completed.

COMPLICATIONS FOLLOWING PARTIAL LARYNGECTOMY

Hemorrhage. The blood supply of the larynx is derived mainly from branches of the superior thyroid and the inferior thyroid arteries. The superior laryngeal branch of the superior thyroid

artery pierces the thyrohyoid membrane in company with the internal laryngeal nerve. The cricothyroid artery, also a branch of the superior thyroid, anastomoses with the inferior laryngeal branch of the inferior thyroid artery, and both then enter the larynx through the cricothyroid membrane.

In partial laryngectomy, bleeding occurs mainly at the upper (thyrohyoid membrane) and lower (cricothyroid membrane) margins of the operative defect. The base of the defect from which the cartilage has been raised by blunt dissection will be found comparatively dry. From the upper posterior margin of the operative defect, bleeding may be quite profuse from the severed superior laryngeal artery. This vessel should be clamped and secured by suture ligature. It is seldom necessary to tie more than one or two of the smaller branches at this point; the remaining ooze may be controlled by temporary tamponage. Öhngren[11] has recommended preliminary ligation of the superior laryngeal artery to prevent post-operative hemorrhage. Although we have not employed it, we believe the procedure to have considerable merit theoretically. Bleeding from the lower margin is seldom profuse, but may be rather troublesome, since the cricothyroid membrane is rather fibrous and clamping of the bleeding point may be difficult.

Post-operative bleeding is best avoided by careful suture ligature of the main bleeding points and patient attention to hemostasis before closing the larynx. Fifteen or twenty minutes may well be spent in gentle tamponage until practically all oozing has ceased. Post-operative bleeding is serious, mainly from the standpoint of pulmonary complications. The actual amount of blood lost is moderate, but its entry into the bronchial tree is apt to result in immediate asphyxia or later bronchopneumonia. The bronchial secretions which are coughed up for the first twelve hours are invariably blood-tinged, but need cause no concern unless the amount and character indicate fresh arterial bleeding. Copious bleeding is an indication for immediate opening of the operative wound and packing of the larynx, after reinsertion of the tracheotomy tube, if it has already been removed.

Subcutaneous emphysema. In three of our patients, the first few coughs after the removal of the tracheotomy tube (6-12 hours after operation) have resulted in moderate subcutaneous em-

physema of the tissues of the neck, due to escape of air into the tissues through the tracheal opening. In one patient, the emphysema, extended as far up as the forehead. As in most cases of subcutaneous emphysema, a moderate febrile reaction occurred, but no other untoward sequel.

Post-operative pneumonia is a complication to be feared in all aged subjects after any operative procedure on the upper respiratory or alimentary tracts. Cancer of the intrinsic larynx suitable to partial laryngectomy occurs most frequently in the aged and the occasional development of post-operative pneumonia cannot be avoided except by excluding from the chance of cure all except the younger more robust patients. Post-operative pulmonary complications seem to follow local as well as general anæsthesia. The avoidance of preliminary medication, especially morphine, and careful attention to hemostasis before wound closure are probably the best prophylactic measures. The cough reflex which should be regained immediately on completion of the closure of the operative wound is a valuable safeguard in expelling the bronchial secretions which have been moderately increased by the stimulation of the anæsthetic and slight oozing from the operative defect. The patient should therefore be placed in bed in the semi-sitting position and encouraged to cough and expel these secretions for the first twenty-four hours.

Granuloma. Redundant edematous granulation tissue may appear in the larynx after four to six weeks and persist for several months. When viewed by a laryngeal mirror, these lesions appear as smooth, pale, often pedunculated masses springing from the unhealed surface, usually nearer the anterior than the posterior commissure. Their appearance is quite characteristic and should cause no great concern. Occasionally, such masses may encroach upon the lumen of the glottis sufficiently so as to cause slight dyspnœa, or to interfere with phonation by preventing approximation of the normal cord. Removal of the granulomatous masses (repeated if necessary) expedites healing and is readily accomplished by a basket punch through the direct laryngoscope. As the raw surface heals, exuberant granulations disappear spontaneously. This complication is most apt to occur when the operative removal extends past the anterior commissure or when a portion of denuded thyroid cartilage is left exposed.

II. Laryngostomy and Implantation of Radon Seeds

This method of treatment is used in our clinic for cancer of the intrinsic larynx too far advanced for partial laryngectomy, because of extension back onto the arytenoids, through the ventricles, deeply into the ventricular bands, or perforation by disease of the cartilage or of the cricothyroid or thyrohyoid membranes.

Perforation is most apt to occur at or near the anterior commissure, since in this region the mucous membrane of the cord in which the growth arises is separated from the cartilage by a very thin layer of submucosal tissues. Further posteriorly, the vocalis muscle forms an intervening layer about one centimeter in thickness. Invasion by disease of the subcutaneous tissues commonly results in the formation of an infected fluctuating tumor at the thyroid notch or just below it. As long as extensive disease remains confined to the laryngeal box, it may be dealt with by total laryngectomy, but once it has invaded the subcutaneous tissues, the situation offers little from the standpoint of radical surgery.

In those cases with perforation, and also in advanced nonperforating disease, we employ laryngostomy and implantation of radon gold seeds. Our present technique has been developed and modified from the procedure devised by Dr. Douglas Quick at the Memorial Hospital over 15 years ago. So far as we know, he first used laryngostomy for exposure in interstitial implantation of laryngeal cancer. The fact that the growth has perforated outside the laryngeal box and produced a subcutaneous abscess seems to be of no particular disadvantage in this method, for such cases offer a surprisingly high percentage of successful results.

Technique of Laryngostomy and Radon Seed Implantation

Local rather than general anæsthesia is always used in these cases, since absolute immobility is not essential. The skin incision is made in the midline from the hyoid to the suprasternal notch. The laryngostomy opening is made exactly as in partial laryngectomy (Fig. 9), except that the cricoid cartilage and upper three or four rings of the trachea are divided as well as the thyroid cartilage. A tracheotomy tube is placed in the lower angle of the wound and a cotton tampon with an attached string is placed just above it to prevent blood from entering the trachea. After

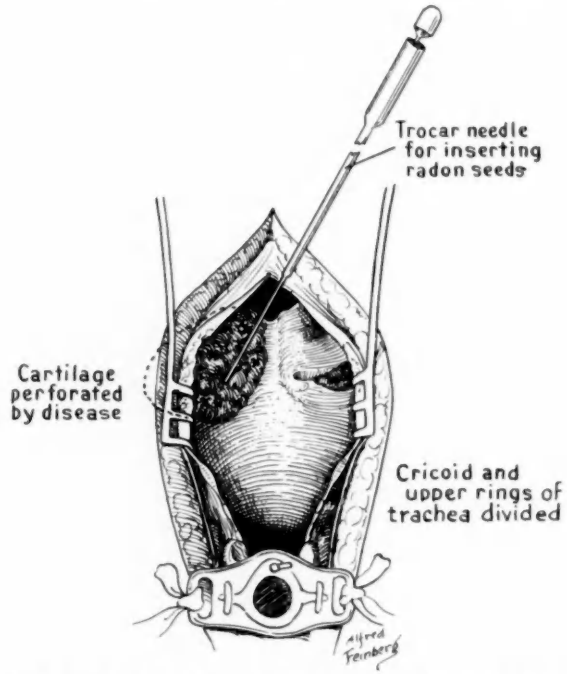

FIG. 9 — (Martin) — Laryngostomy and implantation of radon seeds. The exposure is made exactly as in partial laryngectomy, except that the cricoid cartilage and upper three or four rings of the trachea are also divided. Radon seeds are then implanted in the growth.

careful examination to determine the size and limits of the growth, radon seeds are implanted (Fig. 9) in a quantity sufficient to deliver a tissue dose of about 10 S.E.D. (skin erythma doses) to all parts of the diseased area. In this rather general discussion of the treatment of laryngeal cancer, we can hardly include a detailed discussion of dosage by interstitial implantation or of the best geometrical arrangement of the seeds. We have already published[12, 13] our detailed observations on this subject as they apply to tumors in general.

Considerable experience in the technique of radium therapy in general on the part of the surgeon is essential before he may properly employ such a combination of procedures as laryngostomy and radon seed implantation. Such experience may be gained from the treatment of the more accessible growths of the oral cavity where complications following treatment are less serious.

The individual strength of the seeds should range between 1.50 and 2 millicuries. The total doses in our successful cases have

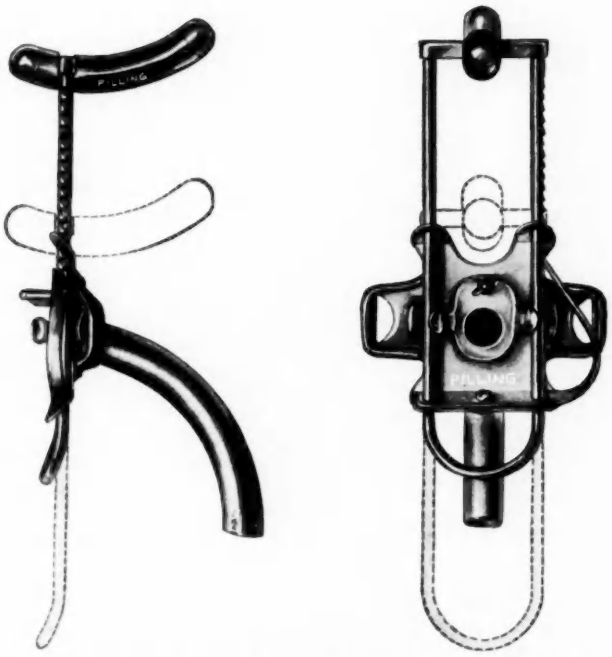

FIG. 10 — (Martin) — Special laryngostomy tube for maintaining the patency of the laryngostomy opening after implantation of radon seeds.

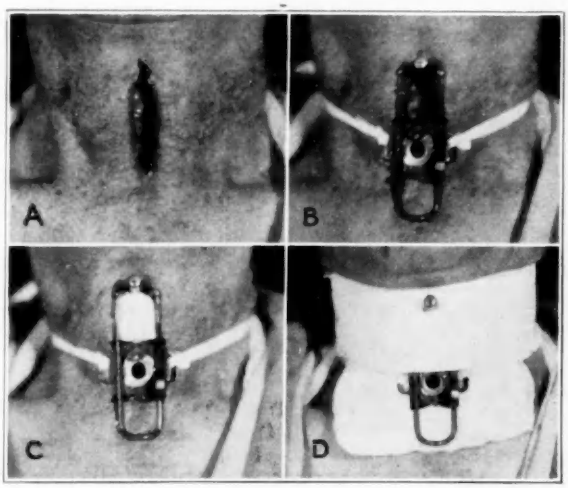

FIG. 11 — (Martin) — (A) Laryngostomy wound about two weeks after operation. (B) Special laryngostomy tube in place. (C) Gauze packing loosely placed between the wound edges. (D) Completed dressing.

varied from 6-40 millicuries, the average dose being between 10 and 20 millicuries. We should advise caution with doses over 25 millicuries, even in the more bulky tumors.

After implantation of the seeds, the tampon and tracheotomy tube are removed and a special laryngostomy tube (Fig. 10) is inserted so as to engage in the upper and lower angles of the wound.* It is usually of advantage to clip off about one centimeter from the anterior portion of each thyroid cartilage either at this time or earlier in the procedure. Plain gauze packing is then loosely placed between the wound edges, care being taken that none of it comes into contact with the tracheal or laryngeal walls, and a dry gauze dressing is placed on the external surface, so as not to cover the opening of the tracheotomy tube (Fig. 11).

After-care and complications. This operative procedure is followed by no more immediate shock or post-operative disability than is simple tracheotomy. The patient should be raised to a sitting position in bed so as to facilitate expulsion of tracheal secretions and drainage through the tube by coughing. Several beds in our wards are equipped to furnish gentle suction from a water syphon. This apparatus functions constantly and quietly. A rubber tube with a glass tip and short rubber catheter leads to the bed so that the attendants (or later the patient himself) may suction off the secretions as they are coughed out of the tube before they can again be drawn back into the trachea during in-

* This special laryngostomy tube was devised by us several years ago, and we have already published its description.[14] It consists of an ordinary Jackson tracheotomy tube to which are attached adjustable uprights to support a saddle, which can thereby be raised and lowered. The instrument is always inserted and removed with the saddle in its lowered position. After insertion, the saddle is raised until it comes into contact with and engages in the upper angle of the laryngostomy wound so as to produce as much dilative tension as is considered necessary to prevent contracture. The sliding uprights are notched so as to permit adjustment as may be required in the individual case.

Without this special laryngostomy tube, we have found that the laryngeal stoma tends to contract very rapidly in the first two or three weeks following operation, so that what remains after about a month is in effect no larger than a tracheotomy opening. In a procedure of this kind, where a large dose of interstitial radium is inserted in the larynx, it is essential that the stoma be kept open so that proper inspection, drainage, and any subsequent treatment may be accomplished through a stoma of adequate size. We have tried out several types of instruments for this purpose. Tucker's laryngostomy tube is of little service in maintaining the patency of the stoma. Baum devised an instrument which consisted of Tucker's laryngostomy tube to which was attached a fixed upright and saddle, but we found the objection to Baum's tube to be the rigidity of the upright and saddle, so that insertion and removal of the device was both traumatizing and painful.

spiration. Suction is also valuable in simple tracheotomies as well as in partial laryngectomies before removal of the tube.

On the day following operation, we usually urge the patient to sit up out of bed. Stereo x-rays of the larynx should be taken at 48-72 hour intervals for the first week to confirm the proper placement and retention of the seeds. If any are extruded, they should be replaced. The first dressing may be postponed for 48 hours, after which the packing and the special laryngostomy tube are removed. A clean tube is reinserted and the packing replaced, and from then on, the tube and dressings are changed daily.

The radium reaction. After about 10-12 days, the growth should show definite signs of regression, accompanied by an active congestion of the parts and the gradual development of a mucositis of the local mucous membranes. The reaction should reach its height, and the tumor should completely regress about 25-30 days following the implantation of the seeds. The height of the reaction is accompanied by a moderately profuse discharge, some local soreness and dysphagia. In the case of bulky, infected tumors requiring large total doses of seeds, localized radionecrosis may develop about 30 days following implantation, and portions of the cartilage may sequestrate and be extruded. The loosened, necrotic tissue should be removed daily by gentle traction and clipping with a scissors. At the height of the reaction, especially if there is much infection and radionecrosis, the patient may complain quite bitterly of pain referred to the mastoid and occipital regions — a common phenomenon in deep laryngeal sepsis. The patient is often found in a characteristic attitude, sitting by the bedside with his forehead resting on the bed. Narcotics, such as acetyl salicylic acid combined with codeine, are required at this time. If the degree of dysphagia prevents swallowing of sufficient nourishment, a nasal-esophageal catheter should be passed.

About 6-8 weeks after the operation, the special laryngostomy tube may be dispensed with and an ordinary tracheotomy tube inserted. The laryngeal stoma if left to itself after this time will not contract down to a smaller size than about 2½ cm. in vertical diameter. The edges heal skin to mucosa and the remaining stoma, about 2-2½ cm. in diameter, will then remain permanently, and the interior of the larynx and trachea may be inspected through it. This defect may be closed quite simply by plastic excision of the healed edges and suture, which we commonly defer for a year or more because of the possibility of recurrence.

In case of extensive disease with marked infiltration and destruction of cartilage, healing may be accompanied by a variable degree of contracture of the lumen at the level of the glottis. If the air space at this point is not adequate for all needs, it may be necessary to leave the tracheotomy tube permanently in place, or at least to leave the laryngeal stoma open permanently. In this event, the end-result is similar to that obtained by total laryngectomy, except that in most cases, an epithelial lined tract persists between the trachea and the pharynx so that the patient is able to whisper or speak with a hoarse voice by simply placing a finger over the opening of the tube or the stoma.

Variations in the procedure. In certain cases where a great bulk of tumor fungates into the larynx or trachea, radon seeds may be inserted at the first stage of the operation and the wound closed by suture, leaving a tracheotomy tube in the lower angle of the wound. About a week or ten days later, the wound is reopened and the special laryngostomy tube inserted, either with or without cautery removal of the fungating portions of the tumor. The wound is then left open and treated as previously described. These variations in procedure have been successful in several instances, and although they have special indications, they also have obvious disadvantages.

III. Endolaryngeal Radiation with the Laryngostat

In certain instances of inoperable intrinsic cancer, the endolaryngeal tissues may be of insufficient bulk to retain securely interstitial implants, as for example, in recurrent disease after radiation or limited surgical removal. In these cases, the application of intracavitary laryngeal radium, if technically possible, offers itself as the best solution of the problem. There have been reported in the past several methods for the endolaryngeal application of radium, usually by inserting the end of a radium-bearing rubber tube through a laryngoscope down as far as the glottis. This procedure is not of much practical significance, since such a device tends to be expelled from the glottis by coughing, despite the use of local and systemic sedatives, so that the duration and the accuracy of the application is uncertain. After laryngostomy, a capsule of radium element may be accurately held in the glottis for an extended period by a device which we have called the laryngostat.

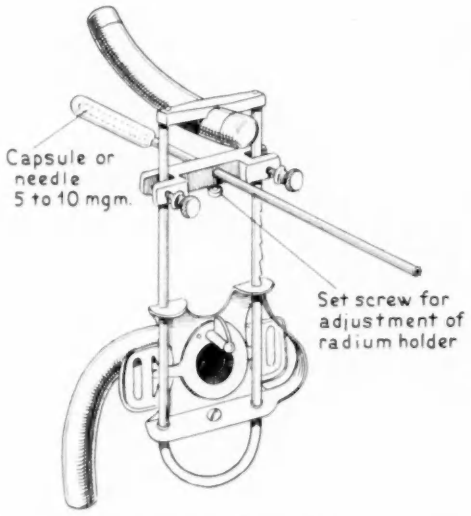

FIG. 12 — (Martin) — The laryngostat — The special laryngostomy tube with an attachment for holding radium.

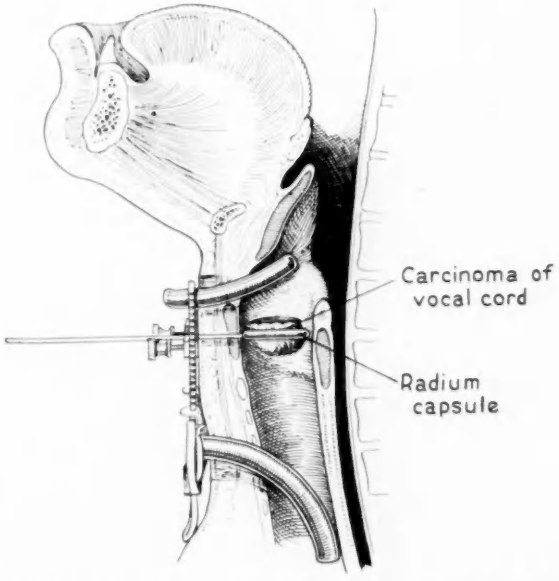

FIG. 13 — (Martin) — The laryngostat — A sagittal view showing the radium capsule being held at the level of the glottis and the tumor.

TECHNIQUE OF ENDOLARYNGEAL RADIATION WITH THE LARYNGOSTAT

After laryngostomy by the technique already described, we insert our special laryngostomy tube for which we have devised an attachment to hold a radium capsule (5-10 mgm.) accurately in the laryngeal cavity at the desired level. The technique is illustrated in Figs. 12, 13 and 14. We recommend a low intensity of radiation protracted over a total treatment time of 3-4 weeks. The procedure is rather new with us, and so far it has been used only in two cases, one of whom has remained well for over a year.* A final decision as to the merits of this procedure must await the results of further trials.

IV. The Treatment of Cancer of the Extrinsic Larynx by the Fractional or Divided Dose Method of External Irradiation

It will be quite generally conceded that surgical extirpation of cancer of the extrinsic larynx has never been a procedure of much practical value. It has probably seldom been successful, except in those cases in which the growth was limited to the posterior surface of the epiglottis or the inner surface of the arytenoids. Within

* About two years ago, there was admitted to our clinic, a middle-aged male complaining of persistent hoarseness, who had been under observation at another clinic for several months with a diagnosis of pachydermia of the larynx. Our clinical examination and two biopsies from the larynx supported this diagnosis. Since we believed the condition to be precancerous, we recommended laryngofissure and prophylactic local excision of the vocal cords. The patient accepted the procedure only after a delay of several months. The operative findings supported the diagnosis of pachydermia, and both cords were therefore locally excised without removal of cartilage, and the larynx was closed. Histologic examination of the excised cords showed diffuse pachydermia with fully developed carcinoma, grade II in one area. The question then arose as to the subsequent management of the case. We believed that the extent of the excision was insufficient to control fully developed cancer. Total laryngectomy might have been performed, but was objectionable from the standpoint of the resultant functional defect. The endolaryngeal tissues were of insufficient bulk to retain interstitial implants. We therefore devised an attachment to our laryngostomy tube by which a capsule of radium element could be securely held at the level of the glottis (Figs. 12, 13 and 14). Five mgms. of radium were used for several hours daily over a period of about 30 days so as to deliver a total dose of 3600 mgm. hours (filter 1 mm. of platinum [equivalent]). The patient has remained free of disease.

In a second case of intrinsic cancer referred to us after protracted x-radiation, we attempted the control of persistent disease by laryngostomy and the application of endolaryngeal radium by the above described technique. As so often occurs when surgery is used following protracted external radiation, the patient succumbed to local complications, mainly radionecrosis, after three months.

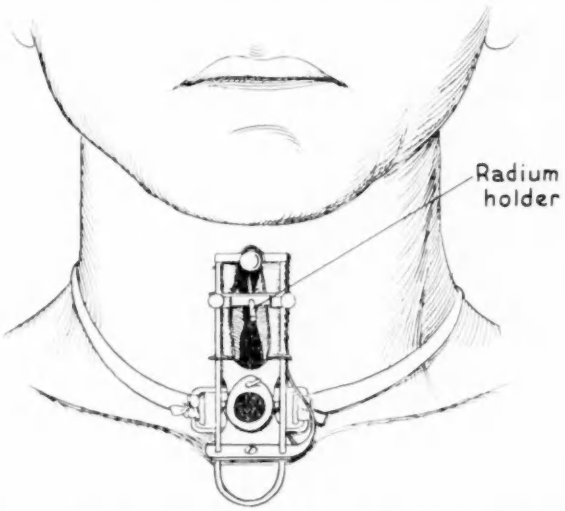

FIG. 14 — (Martin) — The laryngostat in position before the completed dressing.

recent years, with the development of the fractional or divided dose method of external radiation, the results in these cases have been so promising from the standpoint of the control of the primary lesion that, in our opinion, attempts at surgical extirpation or even at the implantation of radon seeds for extrinsic cancer should now be discarded. It is significant that although there are numerous published descriptions of technique for lateral pharyngotomy, laryngopharyngectomy and laryngoscopic excision, etc., the percentage of cures by these methods is almost invariably omitted in the reports.

Dr. Henri Coutard of the Radium Institute of Paris first demonstrated the increased differential effect of the divided dose principle, and first employed the relatively large total doses necessary to obtain complete sterilization of tumors. He proved that for most pharyngeal or laryngeal growths an effective quantity of irradiation given by this method must be such as to be close to the local and general tolerance, in that a marked blistering effect is produced in the skin and mucous membrane directly exposed to the incident beam. He also first demonstrated that such effects produced by the divided dose principle invariably healed without difficulty.

In our opinion, there can be no question as to the greater selective action of protracted irradiation on normal and neoplastic tissues as compared to massive unit doses. The principle of pro-

traction therefore is generally applicable in all forms of cancer except in small superficial easily-controlled growths, as for example, those of the skin, where single massive doses may be preferable as a matter of economy of time, expense and effort. In cancer of the extrinsic larynx, the difficulty of access and the high degree of malignancy make the use of protracted irradiation indispensable for the best results. Before describing the details of the technique, we shall discuss briefly the general principles of protracted x-radiation which apply particularly to this anatomic region, especially in regard to the size, shape and position of the skin portals.

In the earlier reports of Coutard and others (including ourselves), there is little or no discussion as to the significance of the size of the skin portals in relation to the local and general biologic effect. Cancer of the extrinsic larynx tends to metastasize widely in the neck. On the basis of this range of possibility, it is still contended by some observers that the entire node-bearing area of the neck should be included in the skin portal so as to treat all early though impalpable metastases. Theoretically, this principle is sound, but in our experience, it fails from the practical standpoint to attain the desired results for the following reasons. The upper limit of safe dosage with these large portals is insufficient to destroy many of the moderately radioresistant tumors which neverthless are capable of control by the higher tissue doses that may be given safely with smaller portals in smaller volumes of tissue.

In practically all of the earlier work in protracted irradiation of the larynx and pharynx, square or oblong portals of 100-200 sq. cm. were used. In 1931, we, in common with most other observers, employed these large portals which admittedly are of advantage in that slight errors in centering or in the direction of the beam are of only moderate importance. It also is conceded that the depth dose is greater in relation to the intensity on the skin than with smaller portals. With these large ports, the immediate effect on the primary lesion is usually quite promising, but it will be found that the local and general tolerance is exhausted with only a moderate dose, so that in many lesions of average radioresistance, treatment must be discontinued before complete or permanent regression of the growth has occurred. Furthermore, even in those cases which are permanently controlled by the use of these large ports, the late radiation effects in the large volumes of normal tissue are such as to produce a permanent disability, sometimes more uncomfortable and disabling than the original

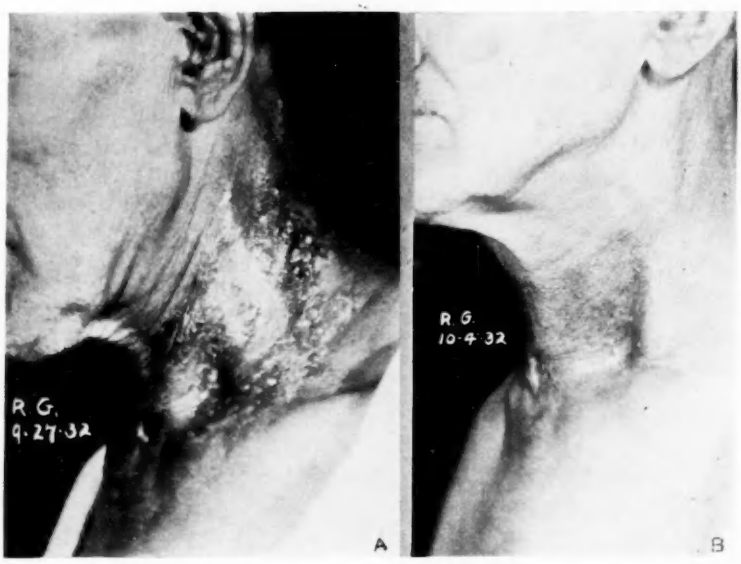

FIG. 15 — (Martin) — (A) The epidermicidal reaction of the skin of the neck immediately following the administration of 3500 r to each side with a portal 7 cm. in diameter. (B) The healed condition one week later.

tumor, although these sequelæ may not in themselves always be lethal. It must be realized that the control of laryngeal cancer by radiation necessitates marked effects in the skin, mucous membranes and other normal tissues from which they eventually may recover to an acceptable degree, provided that these reactions are not too extensive in area and volume. For these reasons, we advise the use of the smallest possible portal which will include the whole volume of the lesion or lesions to be treated. The importance of this principle has also been emphasized by Juul.[15]

From the standpoint of the roentgenologist, the skin reaction has usually been the criterion of safe radiation dosage. From the broader surgical viewpoint, the skin reaction is about third in importance of the biologic effects from the standpoint of overdosage by protracted radiation. In our opinion, there are two biologic effects of more clinical importance than the skin reaction, namely, the general constitutional effect and the deep tissue reaction.

The skin reaction may be directly observed and the tolerance rather accurately determined. Provided that a reasonably sized portal is used, the blistered skin caused by adequate protracted irradiation in laryngeal or pharyngeal cancer always heals, al-

though the degree of the reaction constitutes a surgical lesion in itself which requires surgical management and dressings similar to any second degree thermal burn. In order to cure extrinsic laryngeal cancer by radiation, we must in most cases deliberately produce a surgical lesion in the skin, the eventual success of the procedure then depending on the surgical care of the lesion so produced (Fig. 15). Properly treated, these reactions in the skin constitute no major difficulty. If not properly cared for in the surgical sense, many of the skin reactions which would otherwise heal may go on to necrosis of the superficial layers and persistent ulceration.

With large portals covering the entire side of the neck, there is produced a general constitutional reaction, sometimes to moderate and always to heavy total doses. In such cases, there is a loss of weight and strength and a fall in the leucocyte count before any marked local effect is produced in the skin, mucous membranes or in the tumor. If persisted in, such treatment may bring on a fatal termination, due to the volume of tissue irradiated, rather than to the tissue dose at any point within it. This constitutional effect decreases with a decrease in the size of the skin portal, and the attendant decrease in the volume of tissue irradiated. The tolerance of the organism as a whole depends upon a mutual adjustment of the volume of tissue irradiated (determined by the size of the portal), the tissue dose within the volume (determined by the size of the total dose), and the total treatment period. For example, with portals 10×15 cm. in diameter and daily doses of 400 r, a total dose of 2400 r $\times$ 2 (2400 r to each of two portals) is reached in 12 treatment days, which is about the upper limit of safe dosage with these factors. If one went on to a dose of 3000 r $\times$ 2 or more with these factors, a certain number of fatalities would result from the constitutional effect of the treatment. If, on the other hand, the portal is reduced to 7 cm. in diameter, 350-500 r in daily doses may be continued safely to 4000 r $\times$ 2 or more without undue constitutional effects. The local reactions in the skin and mucous membrane heal without difficulty and the regression of the lesion is more prompt than with the larger portal. The tolerance of the local tissues depends to a considerable extent upon the same factors, but more particularly on the local tissue dose.

With the conviction that the volume of tissue irradiated should be as small and as efficient as possible, we use circular rather than square or oblong portals whenever possible. Only in the case of

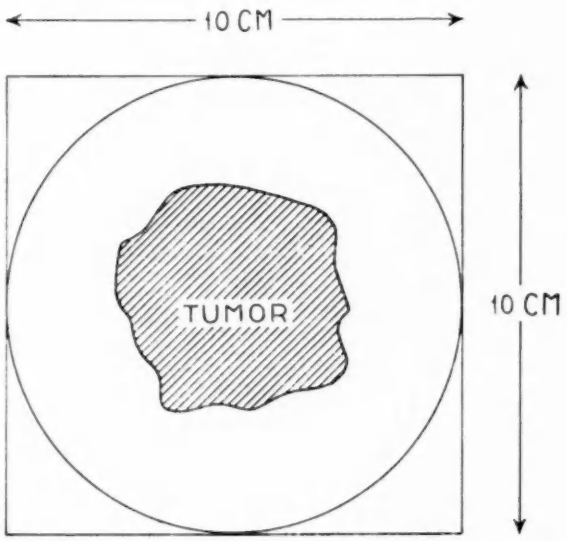

FIG. 16 — (Martin) — Since the edges are equi-distant from the tumor with the square and circular portals of equal diameters, such portals are equally efficient for the treatment of a tumor of the relative size of that indicated in the diagram. However, the surface area of the circular portal is 20 per cent less than the square. Therefore, the local and general tolerance is conserved by the use of circular rather than square portals.

irregularly widespread metastases in the neck can an oblong portal be used to advantage. In Fig. 16, we have represented a theoretical lesion to be irradiated by a square and a circular portal of equal diameters. It will be noted that the surface area of the circular portal, which is efficient, is 20 per cent less than the square. The tissue volume receiving the direct beam is therefore 30 per cent less with the circular portal, which, if equally efficient, should conserve the general tolerance by avoiding unnecessary dosage to outlying normal structures.

We found that if the beam of radiation is limited by a metal cone attached directly to the tube holder, the beam may be accurately centered and directed over the desired tissue volume. We have constructed a series of such cones or cylinders (Fig. 17) varying in size from 4 cm. to 10 cm. in diameter. The smaller cylinders (less than 7 cm. in diameter) are difficult to localize accurately and may become seriously misdirected on the slightest movement of the patient during treatment. Unless there are special indications, we do not use cylinders smaller than 7 cm. in diameter. These cylinders are of practical value for several reasons. Since they are placed in contact with the skin (Fig. 18), the exact localization of the skin portal is more readily accom-

FIG. 17 — (Martin) — Metal cylinders varying in size from 2½ to 15 cm. provide circular skin portals for protracted irradiation of intraoral pharyngeal and laryngeal cancer.

plished from day to day, especially if the skin be marked with a dye. The patient is much less apt to move, since the cylinder placed against the skin tends to steady him, and finally the useful surface area of a circular portal is greater than a square portal.

We believe the essential features of the divided dose principle as first developed by Coutard[16] to be as follows:

(1) The treatments should be given daily (or at least at short intervals), and should be of equal quantity, unless the clinical course indicates a raising or lowering of the daily dose.

(2) A total treatment period of a definite length (15-20-30 days or longer) should be decided upon in which to deliver a certain total dosage. This treatment period should be adhered to unless the clinical course indicates that it should be shortened or lengthened in the individual case.

(3) In most cases of extrinsic laryngeal or pharyngeal cancer, the total biologic effect produced must be close to the local and general tolerance, in that a definite blistering of the skin and a mucositis must be produced in order to approach a lethal effect on most tumors.

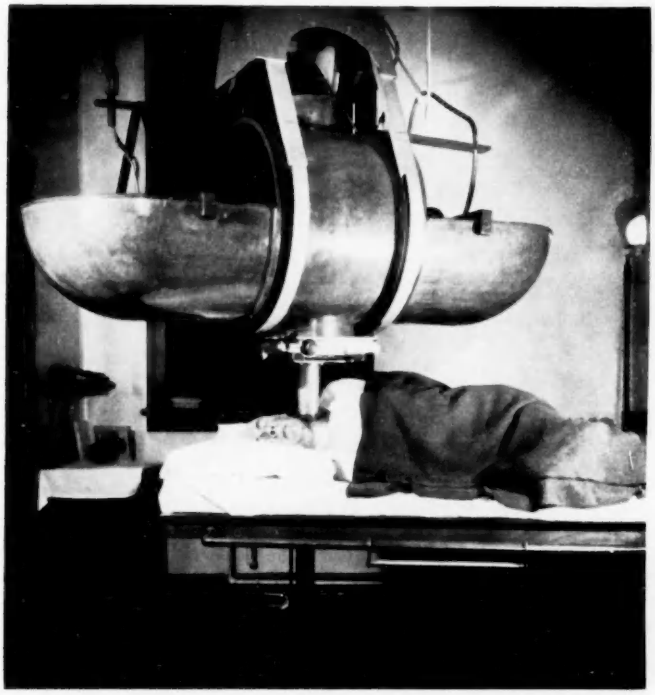

FIG. 18 — (Martin) — Carcinoma of the extrinsic larynx being irradiated through a cylinder 7 cm. in diameter.

The best plan of procedure in the various types of neoplasms in the various localities may not be definitely settled as yet. It is interesting to note that among the published reports, there is general agreement on the broad principles outlined above. There is, however, wide disagreement as to such details as intensity, filter, distance, and frequency of treatments (twice daily, once daily, every two or three days, etc.). We are forced, therefore, to conclude that a wide variation in the latter details is consistent with comparable end-results.

At the present time, we are using mainly roentgen radiation with the following factors: 200 kv. (peak); 30 ma.; 60-65 cm. distance; filter 0.5 mm. Cu + 2.5 mm. Al; circular portals 4-10 cm. in diameter, obtained by the use of metal cylinders of various diameters.

TECHNIQUE OF THE TREATMENT OF CANCER OF THE EXTRINSIC LARYNX BY THE FRACTIONAL OR DIVIDED DOSE METHOD OF EXTERNAL IRRADIATION*

The first requisite in outlining treatment in a given case is the careful localization of the tumor. This we believe is best accomplished by indirect mirror examination. By this means, the whole hypopharynx and larynx is brought under vision so that the extent and limitations of the primary lesion can be accurately determined.

By the help of the palpating finger, the exact situation of the underlying tumor is marked on the skin of the neck by a dye, such as gentian violet or carbolfuchsin. The relationship of any metastasis is then determined so that the skin portal will amply include both primary tumor and metastasis. The size and shape of the skin portal, the direction of the beam, and the daily dose are then decided upon. The center of the skin portal should be localized in relationship to some fixed anatomic landmark (angle of the jaw, hyoid bone, part of the thyroid cartilage, etc.). At the present time, we aim at a treatment period of about 20-25 days, extending it and the total dose if the clinical course and local and general tolerance so indicate.

To insure the correct and uniform centering of the portal at all subsequent treatments, we always mark the position of the primary lesion and the border of the skin portal on the skin with a dye. So far, we have not found an entirely satisfactory indelible dye for this use. Gentian violet and mercurochrome will disappear after washing or shaving. The following formula is of greater permanency, but should be reapplied daily:

Pyrogallic acid	3
Tinct. ferric chloride	5
Acetone	30
Alcohol q. s. ad.	60

A 10 per cent solution of silver nitrate may be safely applied in one spot a few millimeters in diameter, and is more permanent than any of the above mentioned.

* The technique of protracted roentgen therapy in extrinsic laryngeal cancer is almost identical with that used in pharyngeal cancer. Extensive literature on this mode of treatment has developed within recent years to which the reader is referred.[15 to 26]

With circular portals of 7-10 cm. in diameter (40 sq. cm.-60 sq. cm.), we begin with a treatment of 350-400 r (measured in air) daily to alternate sides of the neck, so that at the end of 20 treatments, the patient has received 3500-4000 r to each side.

For a single lesion of moderate size, that is, up to 3 cm. in diameter, we favor the use of the smallest feasible portal. We have chosen 7 cm. as the smallest portal compatible with fairly accurate localization. Smaller portals are used only in special situations. Larger portals are required when the primary and metastases are not directly superimposed. We seldom use portals larger than 10 cm. for the treatment of laryngeal cancer.

The cases are examined daily and any variations from the technique originally decided upon will depend on the clinical course. From the fifteenth to the twentieth day, the patient should be more closely observed, from the standpoint of the intensity of the local reaction, the degree of regression of the tumor, and the general tolerance of the patient. In most cases, the treatment should be continued until there is a blistering radiodermatitis of the skin, and mucositis of the mucous membrane exposed to the direct beam. These reactions are the "epidermite" and "epithelite" of Coutard.

In a small percentage of cases, the treatment must be discontinued before an average dose has been administered, because of the severity of the local reaction, and the lack of general tolerance. In other cases, it will be found that less than the expected reaction has occurred, both in normal and neoplastic tissues. In such cases, the treatment is continued for 2-6 days, giving an additional 300-700 r to either side of the pharynx. We have found that the amount of radiation necessary to produce a given effect, such as blistering of the skin, may vary as much as 25-30 per cent in different individuals.

Using a 20-25 day treatment period and roentgen rays at 200 kv. with the physical factors described above, we have found that 3500 r to each of two portals on either side of the neck is the average safe dose with circular portals 7-8 cm. in diameter. Additional treatment is not given under such conditions, without careful consideration as to the safety and necessity in the given case. We have not found doses of more than 4200 r $\times$ 2 to be productive of good results. In some cases, the tumor will not completely regress, even under such doses, and in these, the method cannot be pushed further with success, in our experience.

If with the same set-up, circular portals of 9-10 cm. or larger are used, the daily doses and total dose would be decreased so as to deliver about 3000 r × 2 in 20-25 days. With smaller circular portals 4-6 cm. in diameter, the doses should be increased to deliver 4000-5000 r × 2 in 20-25 days. In most cases, it is best to err on the side of heavy treatment, since we have found that a marked reaction is usually followed by prompt healing and excellent regression of the tumor.

We see no advantage in deliberately interrupting the treatment so as to give the patient several days of rest before completing the original plan, as is recommended in some cases by Coutard. When unusually severe reactions develop, or in the case of a surgical complication, such as tracheotomy, the daily sequence of treatments must necessarily be temporarily discontinued. However, we favor an uninterrupted sequential series of daily treatments in all cases, if possible. (In our roentgen therapy clinic, no treatments are given on Sundays or holidays). If only one portal is used, we ordinarily give treatments on alternate days, but only as a matter of convenience. It seems to us that a rest period of several days during the treatment period is bound to introduce an unnecessary element of empiricism and uncertainty, which has always been a drawback to scientific radiation therapy. If the necessity for interrupting treatment arises too often, it indicates that the daily doses have been too high.

At about the 20th-25th day, a decision should be made as to the subsequent management of the case. Depending on the clinical findings at this time, one of three possible courses may be followed. First, if there has already been complete regression of all clinical evidence of the disease for several days, the treatment may be discontinued and considered complete. Secondly, further external radiation may be given, that is, after 3500 r × 2 have been administered, the treatment may be continued until the dose has been pushed to 4000 r × 2 or rarely 4500 r × 2 with average portals (7-8 cm. in diameter). In our experience, we have seldom found it of value or even safe to push the dose much beyond 4000 r × 2 to the larynx with the average portal. Thirdly, it may be decided at about the 20th-25th day to discontinue external radiation and to supplement the treatment either in the primary lesion or in the neck nodes by the implantation of radon gold seeds.

Supplementary implantation of radon seeds in the arytenoids or ventricular folds after heavy protracted x-radiation almost

invariably results in local radionecrosis. This complication in the hypopharynx is so serious that we advise against the supplementary use of seeds in these locations. Residual disease in the epiglottis is difficult to implant because of the fragility of the tissues, but the complication of radionecrosis is not so serious at this point.

THE TREATMENT OF CERVICAL METASTASES FROM EXTRINSIC LARYNGEAL CANCER

Cervical metastatic nodes in laryngeal cancer will commonly prove to be more resistant to radiation than the primary lesion, so that even if a neck node lies directly superimposed on the primary lesion, the node commonly persists after the primary has completely regressed clinically. In these cases, we decide at the end of 20-25 days whether or not to implant radon seeds. Implantation may be accomplished through skin punctures, but accuracy in placement is favored by surgical exposure of the outer surface of the node by a short incision, so that seeds may be implanted under direct vision and immediate palpation (Fig. 19).

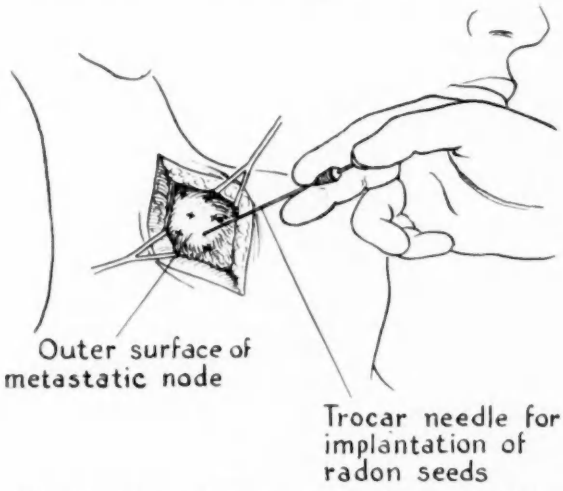

FIG. 19.—(Martin).—Conservative treatment of cervical metastatic lymph nodes. After protracted external radiation, radon gold implants are inserted. These may be inserted through the skin, but accuracy is favored by surgical exposure of the outer surface of the node for implantation.

If the node lies at too great a distance from the level of the primary lesion to be included in the same skin portal, the node in question is irradiated through a separate portal of a diameter at least 2 cm. beyond its borders, and depending on the size of

the portal, protracted irradiation is given daily for 10-15 days (3500-5000 r) and then radon seeds are implanted. By not waiting too long, we anticipate the skin reaction from the x-radiation which comes on after the operative wound has at least partly healed, but if the tissues are handled gently, an incision may be made directly through the blistered skin without undue delay in healing, except that the apparent degree of the reaction is increased by the surgical trauma.

The dosage of radon seeds employed in these cases varies from 6-10 S.E.D. (Table II), depending somewhat on the amount of external radiation already given, the character of the growth and lastly on the size of the node. In small nodes, one may safely overdose and thereby irradiate a questionable volume of tissue outside the node, while in large metastases the local and general tolerance may necessitate giving a minimum tissue dose in order that the total dose may be tolerated.

TABLE II

MILLICURIES IN GOLD SEEDS (0.3 MM. GOLD FILTER) REQUIRED TO DELIVER SPECIFIED DOSES TO MASSES OF VARIOUS DIAMETERS

Skin Erythema Doses	Diameter of Mass — Centimeters											
	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	6.0	7.0	8.0
	Number of Millicuries											
5....	1.0	2.5	4.0	7.5	10	12	14	17	20	27	35	45
6....	1.2	3.0	4.8	9.0	12	14	17	20	24	32	42	54
7....	1.4	3.5	5.6	10	14	17	20	24	28	38	49	63
8....	1.6	4.0	6.4	12	16	19	23	27	32	43	56	72
9....	1.8	4.5	7.2	14	18	22	26	31	36	49	63	81
10....	2.0	5.0	8.0	15	20	24	29	34	40	54	70	90

By this strictly local treatment of clinically palpable nodes, one conserves the local and general tolerance so that if further metastases appear, the local tissues and the general condition of the patient will permit further treatment. When large portals covering the entire node-bearing area are used, the local and general tolerance may be completely exhausted so that adequate treatment of later metastases cannot be given safely.

COMPLICATIONS AND SEQUELÆ

General systemic reactions may occur following heavy protracted external radiation with large fields. Under these circumstances, there often is produced a chronic radiation sickness not due to actual local discomfort, dyspnœa, lack of nutrition or to changes in the skin or mucous membrane. The patient becomes listless, weak, toxic, loses weight and becomes cachectic. Such a condition is probably due to the volume of tissue irradiated rather than to the local intensity of the effect, or possibly to the partial derangement in function of the various glandular, vascular and nervous structures of the neck. Such a complication can be prevented by avoiding the use of unnecessarily large skin portals except when required by the widespread character of the disease. If large portals (200-300 sq. cm.) are used in such cases, the daily doses must be cut down to about 150-200 r, the total treatment period extended for 7-10 days longer, and the total dose limited to 2500-3000 r $\times$ 2. The use of such large portals, whether necessary or not, is always indicative of an unfavorable prognosis from the standpoint of cure.

The acute radiation effect in the skin and mucous membranes directly exposed under the beam of radiation does and should go on in most cases to an acute blistering or epidermicidal reaction (Fig. 15). This degree of skin and mucous membrane reaction is necessary if one is to produce a lethal effect on most extrinsic laryngeal or pharyngeal tumors by this method. The character of the skin change is alarming in appearance to observers unfamiliar with it, but one may be assured that such a reaction produced by repeated treatments over a period of 15-30 days always will heal under proper care without any serious sequelæ. In this regard, it differs from an effect of the same apparent degree caused by one or two heavy doses or that produced in tissues subjected to repeated exposures at long intervals.

The typical skin reaction begins with an erythema after about the first week which goes on to a deeper red, dry, sometimes pigmented appearance. Then the superficial or epidermal layers of the skin become raised by a layer of serum which being denuded leaves the corium as a deep red weeping raw surface. While the skin is in a dry state, frequent applications of vaseline or mineral oil may be used with no other dressing. When the surface is denuded, the affected area should be kept covered with a single layer of fine mesh gauze bandage impregnated with vaseline or

boric acid ointment or the following hygroscopic ointment which we have found efficacious.

Lanolin (hydrated)	2
Zinc oxide ointment	1
Starch	1
Liquid petrolatum q. s.	

The raw surface should be sponged daily with sterile saline to remove the exudate, and the surrounding normal skin cleansed with soap and water and alcohol. If the raw surface is protected from dryness, crusting, trauma and secondary infection, it ordinarily heals completely in 10-14 days, leaving a soft pliable skin in which moderate telangiectasis may or may not later appear.

The mucosal reaction, a counterpart of that in the skin, should go on to a membranous mucositis of diphtheritic appearance in the tissues exposed to the direct beam. Although not painful while at rest, this lesion is quite tender on movement and bleeds slightly but easily if the membrane is disturbed. It reaches its maximum about 20-30 days after the beginning of treatment and usually disappears in about 10-15 days, depending on the size of the total dose. During its height, there is considerable serous and mucoid discharge from its surface, with a diminution in the quantity and an increase in the viscosity of the saliva. If not relieved, the accumulation of these secretions is the cause of a great deal of discomfort.

As a routine at the beginning of treatment, we order throat irrigations every two hours with a two-quart irrigating can with rubber tube and glass nozzle, using a solution of sodium bicarbonate in warm water. At night, the patient is instructed to take the irrigation if he awakens and notes any marked discomfort. This simple procedure affords a marked degree of relief as is proven by the fact that it is voluntarily kept up by the majority of patients without further direction. The viscidity of the secretions is relieved in great part as soon as the mucositis clears, but dryness of the mucous membranes may persist in a lessening degree for several years after treatment, a complication which we shall discuss presently under xerostomia. The mucous membrane reaction is partly responsible for the dysphagia which sometimes occurs.

Dysphagia is always present to some extent when an acute radiation reaction is produced in the pharynx below the level of the soft palate. If due to this cause alone, the disability and

discomfort are of only moderate degree, but even moderate dysphagia may be responsible for a marked loss of weight and strength in a non-cooperative patient. When a reaction is produced in the base of the tongue by radiation directed to the aryepiglottic folds or epiglottis, there is always a temporary loss of taste which in turn causes a loss of appetite. The patient should be instructed as to the necessity for the maintenance of his weight and nutrition and should be encouraged to take an adequate balanced liquid diet of high caloric value. A nasal feeding tube is often a satisfactory means of relief for a few days during the height of the reaction, a procedure which in difficult cases may be accomplished by instrumentation through a direct laryngoscope.

The more serious types of dysphagia are obstructive in character due to the situation of the growth or to swelling and edema in the hypopharynx. In some cases of extrinsic laryngeal cancer, the patients are found to be suffering from marked dysphagia and malnutrition on admission. In the latter, one can seldom depend on radiation treatment for the immediate relief of this difficulty. As a matter of fact, radiation of an extrinsic laryngeal growth will almost invariably cause an immediate though possibly temporary increase in dysphagia. If the dysphagia is due to involvement near the upper end of the esophagus or occurs late in hypopharyngeal cancer as the result of marked lymphedema or radionecrosis with deep infection of uncontrolled cancer, gastrostomy is the only method of relief. In such cases, we prefer a permanent form of gastrostomy such as the Janeway operation,[27] in which there is no danger of spontaneous closure of the stoma and the presence of a tube is not required except at the time of feeding.

Dyspnoea is seldom a marked initial symptom in cancer of the pharyngeal portion of the extrinsic larynx. However, when extrinsic laryngeal cancer is far advanced, or when the hypopharynx has been heavily irradiated, there may be sufficient swelling of the arytenoids or ventricular folds to cause obstruction. During treatment of the hypopharynx by protracted irradiation, one should be on the lookout for even the slightest audible laryngeal stridor, which is indicative of beginning respiratory obstruction. Swelling or edema in this region may progress rapidly and it is far better to perform an unnecessary tracheotomy than to send the patient home and risk a fatal degree of obstruction. Although our available bed space does not permit it, we should

prefer to admit all extrinsic laryngeal cases to the hospital during treatment.

A tracheotomy is disadvantageous during active treatment, since the presence of the tube is inconvenient in accurately centering and applying x-radiation. The tracheotomy wound is a portal of entry for infection into the perilaryngeal tissues whose resistance is already somewhat lowered by the presence of cancer and heavy radiation effects. In spite of these disadvantages, the procedure does not preclude a successful result and should not be withheld when indicated. The incision should be kept as short as is feasible and should be placed as low as possible, just above the sternal notch.

Pulmonary complications in the form of bronchopneumonia or pulmonary abscess are not uncommon in extrinsic laryngeal cancer. In our experience, they are most apt to follow obstruction in the pyriform sinuses or upper end of the esophagus. When obstruction occurs lower down in the esophagus itself, the salivary secretions and other discharges will enter the esophagus to be held in the pouch-like portion just above the stricture. Overaccumulation will bring about regurgitation of this collection into the pharynx to be spat out of the mouth at relatively long intervals. When obstruction occurs just above the upper end of the esophagus or in the pyriform sinuses, there is no reservoir to receive the constant trickle of liquid discharges into the hypopharynx, and as a result, the pyriform sinuses fill so that the excess pours over the notch of the interarytenoid space into the glottis and down into the trachea. This phenomenon we have observed repeatedly. There is, of course, a natural tendency toward the expulsion of this aspirated material by coughing, but even this safeguard is lost during sleep or when infection of the growth and local cellulitis make coughing painful. The necessity for and the presence of a tracheotomy tube in these cases favors such a complication.

Radionecrosis, if not too extensive, is not necessarily a serious complication in the upper portions of the pharynx, but its occurrence in the hypopharynx is always of bad prognostic import. Although most radionecrosis of the deep tissues is the result of heavy interstitial radiation, it may be caused by external radiation alone. By crossfiring two beams of x-radiation in heavy dosage so as to avoid mutual irradiation of the skin portals, it is possible to produce massive radionecrosis in the deep tissues in the midline of the pharynx (extrinsic larynx) with prompt and com-

plete healing of the overlying irradiated skin. This is one of the reasons for our contention that the degree of skin reaction is not a criterion of safe dosage in protracted radiation. However, this complication seldom occurs in the deep tissues as the result of external radiation alone and the subject is therefore of no great practical interest.

When supplementary interstitial radiation is used for the treatment of residual disease after heavy protracted external radiation, one must always consider the possibility of localized radionecrosis. This complication in a small volume is of no serious clinical consequence in the base of the tongue, tonsil, soft palate or upper lateral pharyngeal walls. However, in the hypopharynx (arytenoids and pyriform sinuses), the presence of even slight localized slough is of serious significance, since once initiated, the process commonly pursues a progressive course, extending widely into the perilaryngeal tissues and cartilages to terminate in a combination of esophageal and laryngeal obstruction, sepsis and hemorrhage. Once hypopharyngeal radionecrosis has become well established, we know of no markedly successful measure for relief. Laryngostomy for drainage after heavy external and interstitial radiation of the larynx is of little value.

When radionecrosis occurs in any portion of the pharynx or extrinsic larynx, the patient should be seen and treated daily as long as the complication persists. With a specimen forceps, the slough is gently manipulated and the superficial loosened portions removed and the area irrigated with a power spray. Too aggressive attempts at the withdrawal of slough is not only painful, but is apt to produce hemorrhage. By repeated daily cleansing and conscientious care, the slough may eventually sequestrate, leaving a yellowish, rather avascular, granular base which slowly heals.

Hemorrhage may occur as the result of erosion of one of the larger arterial branches by the disease itself or by radionecrosis and slough. The greatest danger in hemorrhage from the extrinsic larynx is the aspiration of blood into the bronchial tree and asphyxia or bronchopneumonia. Tamponage is not often possible in the hypopharynx. Ligation of the external carotid and its branches (including the superior thyroid in hemorrhage from the extrinsic larynx) may be indicated, but is not nearly so successful as in bleeding from the upper portions of the pharynx or oral cavity.

Xerostomia. After healing of the acute reaction from diffuse irradiation of the extrinsic larynx, there may persist a chronic dryness of the mouth and pharynx. This complication is due to a dysfunction, not only of the major salivary glands (parotid, submaxillary and sublingual), but also of the minor salivary glands in the mucosa of the palate, base of tongue and pharyngeal walls. The secretions of all of these glandular structures contribute to the moistening and lubrication of the oral and pharyngeal mucosa. Chronic xerostomia is most apt to be a troublesome complication following heavy irradiation through large skin portals (100-200 sq. cm.) in which all of the major salivary glands, as well as a large portion of the pharynx are included within the primary beam. By the use of smaller portals (50-75 sq. cm.), whenever possible, the severity of this complication is greatly reduced. We have not found any satisfactory means of relief for chronic radiation xerostomia. The discomfort is temporarily lessened by frequent local applications of liquid petrolatum. Chewing gum seems to stimulate the flow of saliva in some instances. After a year or two, the secretions tend to return to normal.

PALLIATIVE TREATMENT OF THE ADVANCED HOPELESS PATIENT

There is much to be learned from the observation and symptomatic relief of the hopelessly advanced cases. It is natural that one should prefer to treat early favorable cases, but from the purely humanitarian standpoint, palliation of the incurable patient is equally worthy of effort. By treating all patients, no immediate or arbitrary decision need be made in the borderline case. As our knowledge increases and new or improved methods develop, the range of curability in all forms of cancer is gradually being extended. The palliative measures should consist in more than nursing care, and the administration of opiates, although it must be recognized that too aggressive attempts, either surgical or radiological, only tend to increase suffering and shorten life.

Extensive cancer of the extrinsic larynx, if previously untreated, is almost always benefited by protracted x-radiation to the primary lesion or bulky cervical metastases, even in the presence of systemic metastases. However, if the disease recurs or persists locally, after several weeks or months following heavy protracted external radiation, further radiation is usually of little benefit. To repeat the full dose already given is usually disastrous. Bone metastases from extrinsic laryngeal cancer are relieved of pain by local external radiation.

Hopelessly advanced intrinsic cancer offers a difficult problem from the standpoint of palliation. We advise against the use of protracted external radiation in either the attempt at cure or palliation of this form of the disease. In our experience, it has seldom been of apparent benefit, but is often detrimental. Tracheotomy should not be withheld in the presence of audible stridor.

Advanced cancer of either the extrinsic or intrinsic larynx may encroach upon the esophageal entrance and require the passage of a nasal-esophageal catheter or the performance of a gastrostomy.

RESULTS OF TREATMENT

In our opinion, end-results in the treatment of cancer should be calculated only after a five-year survival period. Most of the cases treated by the techniques described in this report have been admitted during the last five years, so that further time must elapse before our statistics are worthy of extensive analysis and record. Our results, both immediate and late, should be considered in light of the fact that *no case of laryngeal cancer is excluded from our clinic because of the advanced stage of the disease*. The following tentative results are given for what they are worth. Since the method of treatment is selected to suit the particular anatomic form and stage of advancement of the disease, the results of the individual methods should not be considered in a comparative sense.

During the last six years, we have performed 16 partial laryngectomies. Three patients have died from post-operative pulmonary complications. In two of these, bronchopneumonia followed moderate post-operative bleeding. We have had no recurrences, and the remaining 13 remain alive and well — one about six years, 2 over five years, 3 over two years, 3 over one year, and 4 less than one year.

Laryngostomy and radon seed implantation have been performed about 32 times. Sixteen patients remain alive and well — 2 over ten years, 7 from five to ten years, 4 from three to five years, and 4 less than three years.

Cancer of the extrinsic larynx is the most frequent anatomic form of the disease in our clinic. The five-year end-results in 31 cases admitted in 1931 is 4 (13 per cent) alive and free of disease. Since 1932, our results have been slightly better (about 20 per cent), but as yet we are not certain whether the ultimate five-year end-results during these later years may not be reduced

by the development of late metastases. The comparatively poor end-results in cancer of the extrinsic larynx are due only in part to our inability to control the primary lesion. This form of the disease produces few symptoms in its early stages and is usually far advanced with widespread cervical metastases on admission. The failure to cure in the majority of instances is due either to uncontrolled cervical metastases or to systemic dissemination of the disease, the latter being invariably incurable.

SUMMARY AND CONCLUSIONS

In this discussion, there is described in detail the several techniques now in use at the Memorial Hospital in the treatment of cancer of the larynx. It is emphasized that no one treatment method is suited to all varieties of this disease, and that often a combination of methods is the best solution of the problem.

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DISCUSSION

DR. GABRIEL TUCKER, Philadelphia: I am sure Dr. Martin's paper has brought to us a very valuable contribution in the treatment of cancer of the larynx.

There were some few details in the paper which he gave to me that he did not mention. One was the explanation of the fact that 75 per cent of the cases seen at his hospital, the cases he was reporting on, were extrinsic and 25 per cent were intrinsic. This might be put up against the report of most laryngologists who are interested in cancer and who say that about 70 per cent of the cases occur intrinsically.

His explanation was that in his cancer hospital the cases came in late. He explained it in this way that in so many of the cases that were extrinsic

the laryngologists ordinarily found the earlier cases and fortunately they operated on a great many of them and cured them before they got to the cancer hospital, but that they could not do it as well there. Many more laryngologists say that these cases can be fixed up early, which is absolutely necessary for their cure, as we all know.

Nearly all laryngologists interested in cancer of the larynx and treatment of it use some method of partial removal during the fissure or total laryngectomy for intrinsic cancer.

There is one special method which necessitates a subdivision of the classification of intrinsic cancer which is well recognized as an anterior intrinsic cancer, that type that originates on the cricoid, on the membranous portion of the cricoid. In that there have been such very remarkable results and cures by so many laryngologists that we thought it would not be out of place to show here, by moving pictures, the technique of this operation for laryngofissure which is used in early cancer of the larynx of this type.

Some of the statistics that have been reported by Jackson, Thompson, Gordon New and others, including my reports, have given as high a rate of cure as 80 per cent in this particular type of laryngeal cancer. Dr. Shielder has had a number of cases which were treated by laryngectomy without recurrence following it and the cure of the case, and his cases, done in conjunction with Dr. Thompson with his own combination of laryngofissure and laryngectomy showed cures of at least 90 per cent.

So many valuable things have been brought up. I desire to confine my discussion to this particular phase of Dr. Martin's paper, and I would like to show a short movie showing this technique of laryngofissure in this type of case.

(Dr. Tucker showed movies illustrating the operation he referred to, speaking at times regarding what was being shown.)

You will find that the prophylactic injection of cocain in the larynx is sufficient to control cough so that the incision can be done quite accurately and successfully.

A wide margin of the mucous membrane is left around the growth. It is swabbed around here and then removed with the growth in the incision.

This patient has had no recurrence to date, which is a period of about five years. This method of treatment was so well established and so many favorable reports from its use have been gotten that we thought, as a matter of emphasis, it would be well to show the technique.

Dr. Joseph C. Beck, Chicago: I am sure that I voice the expression of the majority, at least, of the audience on Dr. Martin's paper. Although it is not as complete as it no doubt will be when published, it was very illuminating as well as interesting.

I wish to state, not so much in criticism as to mention that I failed to see certain things in the copy sent to me, two things, one of which was the statistical side of this question. It did not need to be elaborate, but we are interested and probably will hear some of it in the closing discussion, something in regard to the number of cases that have been operated on by laryngotomy or laryngofissure or by modified technique, or partial laryngectomy, as it was called, and the end-results.

We have heard regarding the two cases. Of course, two cases are not so many from an institution that I feel has so much material. I had great pleasure in visiting the Memorial Hospital and through the kindness of the gentlemen shown everything that one really wishes to have for the cure of cancer, namely, the pathologist, the eminent Professor Ewing, the surgeons, the radiologists, and the radiumologists.

There is a second point that I wish to make, and that is that I did not hear anything mentioned regarding interstitial irradiation. We are interested, at least we are in Chicago, both at Michael Reese Hospital and at the Veterans' Hospital where there are a large number of cases by this time in the tumor clinics. I had the pleasure of seeing them and watching the work by the radiologic department as well as the surgical department. One knows something about the distant treatment by means of radium. We realize that there is not enough radium in the United States to treat all the cancer about the body by this method, but it is interesting to note that there is an advantage in the distant treatment with radium.

If you remember the remarks made by Coutard himself, in his protracted treatment of carcinoma of the larynx, or carcinoma anywhere, he was imitating radium when he was using the Coutard treatment by x-rays.

So, if we can have radium enough — say five grams of radium pack or balm, as they call it, and treat these cases by that method, I think it is of great advantage.

I have nothing to find exception to in the classification as to the origin of the lesion, or that we should speak of just intrinsic cancer of the larynx and the method, the means, that Dr. Martin has shown should be classified; we are interested when the cases come to us both from the pathological standpoint, and we also want to know what type of lesion it is. That is important, to be sure, but also when the case comes to us it is often difficult, most of the time in fact, to say just where it originated. It is there that we are interested in knowing what type of treatment will apply at that time.

Cancer of the larynx can be cured, we know, in many, many cases positively and remain cured by laryngectomy. The life of the patient is at stake and we are interested in saving the life of the patient. But, from my experience in this work I feel that when I have saved the life of a patient by laryngectomy I have also established, most of the time, an individual who is a handicap to society and is not himself a very happy individual.

We can and we should get the cases early enough. We have preached long enough now. Methods of examination are much easier and definitions of the lesions are established. We should be able to do a laryngofissure and a removal of the cancer without, I think, in most instances, the doing of a heavy laryngectomy, for that is what the procedure really amounts to especially where irradiation, interstitial irradiation, or x-ray will prevent a loss of function of the cartilage. What of that if we have cured the patient and there is enough of an airway, or even with a tracheotomy if there is a way that a patient may talk, and if he can talk through a thin fistula we prefer that to the most perfect laryngectomy that can be performed.

I think that is the crux of the situation as it was shown here today, whether you use a laryngostomy and subsequently let it close up and use

even a tracheal tube. That does not matter. I think that the paper is very constructive and Dr. Tucker's picture and notes that he made were very clear and that we should go away from this meeting feeling that laryngotomy, or laryngofissure and not laryngectomy is the operation of choice.

As radiologists and radiumologists, we must depend on these gentlemen who are in those fields as to what is the best thing for us to do, as to the dosage, the screenage, and so forth.

There is one thing that I wish to say in connection with an anesthetic, and that is that these patients are many times debilitated. They have suffered from obstruction to breathing. They have lacked enough oxygen and they must be prepared thoroughly. We know it is going to be done. But, particularly regarding the anesthetic, the rectal anesthesia by avertin is a great advantage. But, the patient must be studied very carefully before that means of anesthesia is employed. There is nothing better than to avoid the movement of the larynx. So far as local anesthesia of the larynx is concerned, the patient should not cough from irritation of the lower respiratory tract and trachea and bronchi. To have a patient quiet, give an avertin anesthesia and local infiltration, which is always a great advantage.

Dr. John L. Myers, Kansas City, Mo.: I want to ask a question of Dr. Martin. Dr. Beck spoke of there not being radium enough in the country to treat all cancers. Those of you who are members of the American Association for the Advancement of Science were impressed recently to read that x-ray machines are being made today which will produce a gamma ray.

For instance, I think it is in St. Paul where a machine is being made that will deliver 1,200,000 volts and produce the gamma ray equivalent to $100,000,000 worth of radium, and four patients can be treated at a time.

If such can be used we will be able to accomplish what Dr. Beck has spoken of, treating these patients and curing them and leaving a larynx that they can use and so aid the patients that they will not be embarrassed by the operation, as in laryngectomy.

I would like to have Dr. Martin tell us something about that.

Dr. Hayes E. Martin, closing: I wish to thank Dr. Tucker for showing his movie of a partial laryngectomy, which gives a very excellent demonstration of his operation.

Dr. Beck has asked about end-results. Due to the limited time available today, I thought it best to omit a discussion of end-results in the abstract of the paper read today. However, these will be included in the published report. Since you have specifically asked for them, I shall briefly mention the figures. I do not feel that any statement of end-results is worth while, except in regard to cases which have been followed for at least five years, and again a statement of end-results would have to take into consideration the various anatomic forms of laryngeal cancer and not the group as a whole.

First, as to our partial laryngectomies. We have performed the operation in 16 cases which date from about 3 months to 8 years ago. Most of

them have been operated upon within the last five years, so that obviously we cannot speak of end-results. We have had 3 post-operative deaths from pulmonary complications, but no recurrences, and 13 patients are alive and well.

In the extrinsic laryngeal cases, we have obtained about the same results by protracted external radiation as have been obtained in most other clinics. About 20 per cent of cures is about all that one can hope to obtain in these extrinsic cases. This form of the disease is more malignant than the intrinsic form, and even though one cures the local lesion, the real difficulty is that of distant metastases — metastases to the lung or liver, etc.

We have performed laryngostomy and implantation of seeds in about 30 cases, and about half are now alive and well, but again these cases date from a few months ago back to 12 years ago. Again we cannot present five-year results in the whole group, since most of the cases occurred within the last 5 years.

I agree entirely with Dr. Beck concerning the percentage of cures which may be obtained by partial laryngectomy. As I mentioned in the paper, there is no procedure in a serious form of cancer which is attended by a higher degree of success. We believe that partial laryngectomy in early cancer of the intrinsic larynx is the method of choice. However, this operation is not suitable in the majority of cases of cancer of the larynx as we see them in our clinic.

Both Dr. Beck and Dr. Myers spoke of the use of radium in the treatment of cancer of the larynx. I take it to mean that they refer to radium used externally, that is, in the form of radium packs. The alleged superiority of external radiation from a radium pack over x-ray has often been emphasized and publicized. It is sometimes claimed that radiations from radium are superior to those from x-ray. That statement is, we believe, not borne out as far as their comparative value in the treatment of cancer at this time is concerned.

Practically all of the cures in cancer of the larynx by external radiation have been obtained by the garden variety of 200 kilovolt x-ray machines, of which there are thousands in operation in this country. No special apparatus is required. There are many x-ray machines of 400 kilovolts, but at such high voltages, the apparatus becomes rather inflexible and proper adjustment of the tube in relation to the patient's neck is difficult or impossible. It is possible that in the future these difficulties with super-voltage x-rays will be overcome, but at this time, we believe that 200 kilovolt x-rays are superior to any form of radiation from the practical standpoint in curing cancer. The 1,000,000 volt x-ray machines are absolutely rigid as far as any adjustment is concerned, and the patient must be adjusted to them rather than the machine to the patient. These details are of extreme importance in the finer technique of therapeutic x-ray.

Finally, let us consider any supposed advantage in the use of radium rather than x-rays in external radiation of laryngeal cancer. Let us take a concrete example. Suppose that one had four grams of radium element in a pack, and that one gave a daily dose to the larynx at a distance of 10 cm. so as to produce the required biologic effect in 20 days. With this amount of radium at a distance of 10 cm., the patient would have to remain under the radium pack for about 2½ hours daily (10,000 mgm. hours). The

10 cm. distance is rather short for deep therapy, and we are able to use x-rays at 65, 70 or 100 cm. and still deliver the required daily dose under such a plan of treatment in 8-10 minutes, as compared to 2½ hours by radium at the short distance of 10 cm.

If we were to increase the distance with this amount of radium to 15 cm., it would take 5 hours a day to give a comparable dose which one could get in 8 minutes with the ordinary 200 kilovolt x-ray machine at 30 ma. Obviously, even an exposure of 2½ hours in a fixed position daily is far beyond the limit of tolerance of many patients with laryngeal cancer, and, lastly, consider that a four gram radium element pack is now worth in the neighborhood of $200,000 and that with no time out for changing patients, running 24 hours a day, such an apparatus could treat only 9 patients a day, while with a 200 kilovolt x-ray machine costing $4,000, one could treat one patient every 8-10 minutes, or about 80-100 patients a day, at one-fiftieth of the original cost of four grams of radium.

BLOOD TRANSFUSIONS IN DISEASES OF EAR, NOSE AND THROAT

JOHN J. SHEA, M.D.

MEMPHIS, TENN.

A SCIENTIFICALLY administered transfusion is the nearest approach to a perfect tonic available in modern medicine. Once used as a futile gesture in the final fight against a hopeless situation, today a blood transfusion is a valuable therapeutic measure in the pre-operative and post-operative management of promising cases.

The transferring of blood from the donor to the patient is more than a simple intravenous medication and the potential dangers should modify our judgment in the selection of the time and frequency of its administration. If possible, transfusions should be given by surgeons, who confine their efforts to this specialty or who through experience are apt in its administration. The indications for a transfusion in our specialty are:

Hemorrhage of any type, spontaneous, accidental or operative, that acutely reduces the hemoglobin to 75%. The clinical symptom of importance being air hunger, due to the loss of oxygen carrying ability of the blood. Repeated minor hemorrhages over a protracted period or a continued destruction of blood by toxins or poisons to a poverty of 50% hemoglobin demands a transfusion prior to surgery.

CASE I

J. B., age 4, was operated on for the removal of tonsils and adenoid at 8 a.m., May 6, 1935. The child reacted favorably from the operation. At 9:30 a.m. she vomited two ounces of dark bloody stomach contents and a similar quantity at 1 p.m. After a short nap at 4:30 p.m., the child awakened and vomited approximately sixteen ounces of stomach contents containing bright blood. Three minims of Surgical Pituitrin were given hypodermatically every fifteen minutes for three doses in an attempt to control the hemorrhage from the adenoid. Again at 6 p.m. the child emptied her

stomach of seven ounces of fluid containing dark blood. The child became very restless and showed signs of air hunger. Examination of the throat revealed a large clot in the adenoid region with a show of blood in the lower half of the right tonsil fossa. Under general anesthesia, a post nasal tampon was inserted and a vein was ligated in the lower part of the right tonsil fossa. Blood count — Hemoglobin 70%, R.B.C. 2,800,000. The child was of type four and a compatible donor was tested for a transfusion. The multiple syringe method of Lindemann was the procedure of choice and at 9:30 p.m. 120 cc. of whole blood was administered. The child showed immediate relief of the air hunger. After an hour's watching to determine the thoroughness of the control of the hemorrhage, she was allowed to go to sleep. Her night's rest consisted of short naps, but there was no further bleeding or symptoms indicating shock. The post nasal plug was removed fourteen hours after its insertion and the child made an uneventful recovery.

CASE II

J.C., age 5, referred for the removal of tonsils and adenoid on June 18, 1934, with the history of scarlet fever the past March, since which time she continued to have recurrent attacks of tonsillitis complicated by an acute purulent sinusitis.

Examination — The nasal cavity contained a muco-purulent discharge and the tonsils were of the active glandular type. A roentgenological study revealed a persistent infantile type of sinuses with a cloudiness (grade 3) of the antra. Blood count — Hemoglobin 60% and R.B.C. 3,200,000. The child was hospitalized and daily nasal treatments instituted with slight improvement of the character of the nasal discharge. On June 27, one week after admission, the child was given 150 cc. of whole blood by the multiple syringe method. The tonsils and adenoid were removed and the antra drained on June 29. The only complication being an excess of the sanguineous quality of the discharge from the sinuses.

Sepsis — Irrespective of the blood picture a transfusion is indicated whenever a septic patient is not doing well. That demand may require a daily transfusion or at intervals up to five days. Compatible red blood cells live and function as long as 83 days, but the serological improvement is transient and of little value after the fifth day. The symptoms and signs of infection, such as chills, fever and depression are better indicators than the white

blood count with its differential or the red blood count with its hemoglobin content. The latter is usually low as there is a secondary anemia in both the acute and chronic septic patient. Part of the improvement that follows a transfusion is the result of the improvement of the secondary anemia.

CASE III

R.M., age 7, referred June 24, 1935, for a mastoid operation with a history of an acute middle ear for the past three weeks. Temperature on admission was 104, pulse 116, blood count — Hemoglobin 82%, R.B.C. 4,560,000, W.B.C. 13,150, small lymphocytes 16%, neutrophiles 83% (juvenile 5%, stabs 15%, segmented 63%) and basophiles 1%. The child was profoundly ill and looked the part. In spite of the average red blood count and hemoglobin a transfusion was indicated and given with beneficial results. The following day, the mastoid was operated upon. The post-operative convalescence was stormy with a continuation of the septic temperature. On the third post-operative day, the child complained of headache and vomited his supper. Blood count — Hemoglobin 92%, R.B.C. 4,840,000, W.B.C. 16,050, small lymphocytes 22%, neutrophiles 78% (juveniles 5%, stabs 45%, segmented 28%).

Fourth post-operative day — Developed an abdominal pain, which required a consultation with the surgeon to eliminate an acute appendicitis. Blood count — Hemoglobin 87%, R.B.C. 4,500,000, W.B.C. 12,000, small lymphocytes 27%, neutrophiles 73% (juveniles 3%, stabs 13%, segmented 57%).

Fifth post-operative day — Pain over right eye more constant and severe. Blood count — Hemoglobin 86%, R.B.C. 4,500,000, W.B.C. 9,800, small lymphocytes 23%, neutrophiles 77% (juveniles 3%, stabs 8%, segmented 66%).

Sixth post-operative day — Surgeon satisfied that abdominal pain was not from a surgical appendicitis. Blood count — Hemoglobin 80%, R.B.C. 4,160,000, W.B.C. 16,400, polynuclear 71%, small lymphocytes 28%, eosinophiles 1%.

Seventh post-operative day — Nauseated, but did not vomit. Less pain in head. Wound still draining profusely.

Ninth post-operative day — Up in wheel chair. Temperature 99°. Wound only discharging through rubber tube drain.

Twelfth post-operative day — Second transfusion of 300 cc. of whole blood. Wound still discharging more than average. A

mild headache at times during the day. Discharged from the hospital two days later greatly improved.

This child presented a true Gradenigo's syndrome, beginning with paralysis on the twenty-first day and lasting until the thirty-ninth post-operative day.

COMMENT — A blood transfusion is indicated as frequently in a septic case as the surgeon considers his patient not doing well, irrespective of the blood count.

Diseases associated with a decrease in the number of blood platelets (idiopathic purpura hemorrhagica and symptomatic purpura hemorrhagica) produce bleeding, either spontaneous or surgically. Occasionally an unfortunate operation is performed for what appears to be a chronic tonsillitis and a delayed study to ascertain the cause of the hemorrhage results in the finding of a blood dyscrasia. The life cycle of platelets is less than five days, thereby requiring frequent transfusions for this blood disaster. Occasionally preliminary venesection is demanded before subsequent transfusion to prevent plethora.

Hemophilia may be either true or transient. Large transfusions are necessary to supply sufficient platelets or the humoral elements (fibrinogen, calcium or prothrombin) when surgery is demanded or uncontrollable hemorrhages have followed accidents or surgery.

Agranulocytosis — Repeated transfusions have been employed as a means of supplying leukocytes until hemopoietic stimulants have had an opportunity to increase the number of white cells. In contrast, transfusions are of little value in supplying leukocytes during acute infections.

METHODS

The blood may be either transferred in a natural state or modified by the addition of sodium citrate.

Direct Anastomosis of the vein of the donor to the recipient is a very technical operation and difficult to control. No method is available of measuring the volume of blood transmitted and the only practical test is a clinical observation of the improvement of the patient.

Modified Blood — By the addition of citrate to a known quantity of freshly drawn blood, a fluid easily handled and capable of

transportation is obtained. The introduction into the recipient is simple and can be stopped at the onset of any undesirable symptom. Many surgeons prefer this method, claiming for it greater ability to increase coagulation whereas others condemn it as an unscientific soup, devoid of those antigens so valuable in unmodified blood. There are more reactions following the use of citrated than of whole blood. Some surgeons welcome a slight reaction in both the patient with a hemorrhage and septic case.

Unmodified or Whole Blood — Many varieties of apparatus have been presented with claims of their simplicity, but as yet the multiple syringe method of Lindemann remains the safest and most expedient method.

TYPING

To Jansky and Moss belongs the credit of classifying human blood into groups, but we must depend upon the interpretation of the well trained laboratory technician to see that the blood of our donor shall blend with that of our patient. There are four groups of human blood and in order to simplify their designation the National Research Council renamed the groups with letters to avoid the confusion produced by the different numbers used in the two older classifications.

OFFICIAL	JANSKY	MOSS
O	I	IV
A	II	II
B	III	III
AB	IV	I

A safe transfusion is dependent upon the presence in the blood of agglutinins and agglutinogens, the former distributed in the serum and the latter contained in the blood cells. "These substances are not all present in the blood of each individual, but occur in such a manner that it is possible to place almost every human being into one of four groups, according to the presence or absence of the specific agglutinins or agglutinogens. For purposes of orderly thinking the agglutinogens may be conceived of as of more prominence than the agglutinins and are usually designated by capital letters."

Group Equivalent			Group Content and Interaction	
Jansky	*Moss*	*Landsteiner*	SERUM	CELLS
I	IV	O	Contains agglutinins A and B. Agglutinates cells of other three groups.	Contains no agglutinogen. Not agglutinated by any serum.
II	II	A	Contains agglutinin B. Agglutinates cells of Jansky III and IV. Moss I and III. Landsteiner B and AB.	Contains agglutinogen A. Agglutinated by sera: Jansky I and III. Moss III and IV. Landsteiner O and B.
III	III	B	Contains agglutinin A. Agglutinates cells of Jansky II and IV. Moss I and II. Landsteiner A and AB.	Contains agglutinogen B. Agglutinated by sera: Jansky I and II. Moss II and IV. Landsteiner O and A.
IV	I	AB	Contains no agglutinin. Agglutinates cells of no group.	Contains agglutinogens A and B. Agglutinated by sera of other three groups.

SELECTION OF DONOR

A great responsibility should be appreciated by the laboratory technician and the surgeon. The blood of the donor must blend with that of the patient, for incompatible blood may result in death. The donor should be free of syphilis, malaria, asthma, or any allergic sensitivity, and be rich in hemoglobin. In certain instances, it is helpful if the donor's blood contains an abundance of immune bodies. The donor should be young, for the blood of the aged is poorer of antigens. It is not only necessary that the donor and the recipient be of the same group, but their blood should be cross-matched, and the time consumed in this matching is well spent. When the same donor is used for subsequent transfusions, this vital procedure should precede each giving to the patient. The donor never changes his group, but circumstances, especially infection, may produce incompatibility.

REACTIONS

Reactions may be manifested by a chill, rigor, fever, nausea, dyspenia, cough or headache and may terminate in death. A 100 cc. of poorly matched blood may produce a reaction with a hemolysis from which the patient could recover, whereas an unfortunate administration of 500 cc. would produce death. Jaundice and hematuria are evidence of hemolysis, and embolism indicates massive agglutination.

Stetson divides the reactions into (1) Hemolytic, (2) Proteolytic.

The former are the result of poor matching or from using unmatched blood, or because the recipient is suffering from a disease in which an active hemolytic agent is breaking down the cells.

The Proteolytic class embraces three distinct types:

Febrile (the protein contents of the donor's blood influences the type of reaction).

Protein (the dermal reaction of true allergic phenomena with or without systemic reaction).

Anaphylactoid (some of the reactions are of the true protein type and possibly some due to minor agglutinins).

CONCLUSIONS

Transfusion of human blood, a life saving measure, is one of the advances of modern surgery, giving to the patient on demand that which he cannot instantly supply himself.

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DISCUSSION

DR. R. E. STETSON, New York: It is an honor and a privilege to take part in this program of the American Academy of Ophthalmology and Otolaryngology, and it is an added pleasure that my part is to discuss the very excellent paper of my old friend, Dr. John J. Shea, whom I have known since our interne days at Bellevue.

Dr. Shea's paper is remarkable for its scope and conciseness. I can take exception to nothing that he has said. I might elaborate on many points but I believe it would be of more interest and of more value to this audience if I simply supplement Dr. Shea's remarks with a brief résumé of my own clinical experience with blood transfusion in the field of Otolaryngology.

In another fifteen months I will have been intimately associated with this work for a quarter of a century, and for the past seventeen years my entire time has been devoted to blood transfusion, but the time available

is necessarily limited, so that I have confined this report to work done within the past ten years.

About one-third of the work was done at the New York Eye, Ear, Nose and Throat Infirmary at Second Avenue and Thirteenth Street. The excellence of our results there is in no small measure due to the recognition by the attending staff of the value of blood transfusion as a supportive and therapeutic adjunct to surgery and infection. I deeply appreciate the courteous cooperation they have always shown during the many years of my association there.

In reviewing the past decade, I was able to find accurate records of 151 cases requiring blood transfusion for varying conditions within the fields of otolaryngology. Two hundred ninety-seven transfusions were performed on these cases, from 1 to 12 transfusions being required in the individual case and from 100 cc. of blood in infants upon to 1000 cc. in some hemorrhage cases being transfused at one time.

I have divided these cases into four broad groups according to the condition which we were trying to combat.

First is the preoperative and postoperative group of patients. These patients were suffering from a variety of ailments all needing surgical correction. Many of them had an associated secondary anemia. Some suffered from inanition so that they appeared to be rather poor surgical risks and some were simply a lot sicker and more toxic than the average patient. There were 69 cases in this group on which 92 transfusions were performed. The great majority of them required only one transfusion. They all did reasonably well and there were no deaths in this group. Perhaps there would have been no deaths if none of them had been transfused.

The results here are not dramatic, but they are very, very satisfactory. Many a poor risk is quickly transformed into a good one and the surgeon proceeds with his work with much less anxiety. Many a case doing poorly after operation and apparently threatened with a prolonged convalescence shows remarkable improvement after one or two transfusions. Dirty, sloughing wounds are stimulated and show healthy granulations a few hours after transfusion and a toxic temperature often drops to a normal level.

In spite of the satisfactory results, it is in this field that the measure is most often neglected. It is natural to wish to save one's patient any unnecessary expense, but, as a matter of fact, the shortening of their hospitalization will often more than offset the extra expense of a transfusion.

The second group, comprising the hemorrhage cases, while more dramatic, need little comment. The hemorrhage, if severe, requires emergency action; and if slower but persistent, the need for transfusion is quite as evident.

There were 27 hemorrhage cases on which we performed 48 transfusions.

One of the most persistent bleeders was an Italian boy recently brought into the New York Eye and Ear Infirmary following what was probably an operation for septum deviation. We never did get an exact account of the procedure which was carried out in the office of a private physician not connected with the hospital, but it required six transfusions to get him straightened out. No history of hemophilia could be obtained.

There was one death in this group. This was a postoperative A. and T. case which started to bleed about ten days following operation. He was a young man about 20 years of age in excellent physical condition. He bled from both tonsillar fossæ and repeated transfusions and all other measures failed to check the bleeding. We could get no history of hemophilia in this case.

The third and fourth groups comprise those cases where sepsis was a complicating factor.

The third group includes those cases which were definitely septic but in which no positive blood cultures were obtained. There were 34 of these cases on which we performed 77 transfusions. The record of 30 recoveries and only 4 deaths, or a mortality of a little less than 12% is remarkably good. Two of the four died of abscess of the brain. One of the others was moribund at the time of the transfusion.

The fourth group is made up of those cases in which one or more blood cultures were positive so that the infecting organism could be identified. In each of these cases the organism proved to be a hemolytic streptococcus. While undoubtedly this is the most common offender in infections of the mastoid and sinuses, it is probable that some of the failures to obtain positive cultures in the third group was because their infecting organism might have been more difficult to grow on the usual media employed. On the other hand, many undoubtedly had only a transitory bacteremia or else the infecting organism was of lower virulence than in those cases which showed positive cultures.

We need not be ashamed of a record of 7 deaths out of 21 cases or a mortality of only 33⅓%. One of the cases included here was moribund and died before more than one tranfusion could be given. Eighty transfusions were performed on these 21 cases, from 1 to 12 being required in the individual case.

You all know, of course, that multiple transfusions have proved most effective in combating sepsis. Whenever possible I like to repeat every 48 hours until the blood cultures become negative and the clinical improvement warrants less drastic treatment. Such frequent repetition makes it very often desirable to withdraw from ½ to ⅔ of the amount to be given so that amounts up to 500, 600, or even 800 cc. of blood may be given at each transfusion. I am aware that some men advocate the use of much smaller amounts but in my opinion the method described will give better results in some cases.

In conclusion, I wish to present very briefly one of the most remarkable cases of recovery from septicemia that I have ever seen.

Case W. G., 10 year old boy, white, of good family. Admitted to New York Eye and Ear Infirmary on June 4, 1926, with diagnosis of acute mastoiditis of the right side.

Operated on the next day and diagnosis confirmed. Ligation and resection of external jugular was also done at this time as blood culture was positive for streptococcus hemolyticus. A blood transfusion of 500 cc. of whole blood was given following the operation.

In spite of these measures he failed to improve and blood transfusions were repeated on June 8, 10, 12, and 16, 200 to 300 cc. of blood being re-

moved and 500 cc. given on each occasion. During this time he complained of pains in the limbs, particularly the left arm and right leg.

On June 17th, incision and drainage of abscesses of left forearm and elbow were performed and on June 26th the right thigh was aspirated but no pus was found.

The following day, June 27th, a deep abscess of the right thigh was opened and drained and also an abscess on the left ankle.

Transfusions of 500 cc. of blood without any withdrawal of blood were given on June 21st, 26th, 29th, and July 2nd.

For several days increasing pain, tenderness, and size of the liver had been noted and on July 3rd an exploratory laparotomy was performed. The liver was found to extend down to the iliac crest. Exploration with needle failed to find any pus. There was no evidence of peritonitis so the wound was closed without drainage.

On July 5th his condition was very critical — temperature 104°, pulse 140 — but he responded to stimulants and rallied. A blood count on that day showed hemoglobin 90%, R.B.C. 5,840,000, W.B.C. 13,700, polynuclears 84%.

He continued to complain of the right hip and on July 7th the diagnosis of suppurative arthritis of that joint was made. Incision and drainage of the right hip posteriorly was carried out that day.

On July 9th, 200 cc. of blood was withdrawn and 500 cc. given.

On the 15th, spontaneous dislocation of right hip occurred so that it was necessary to apply traction to right leg. Also at this time abscesses of neck and of right groin were incised and drained.

On July 19th, 300 cc. of blood was given without any withdrawal.

On the 20th, and again on the 29th, it was necessary to drain abscesses on the left leg.

The last transfusion of 500 cc. of blood was given on July 31st. His condition was now steadily improving and on August 4th he was transferred to the Orthopedic Hospital and eventually made a complete recovery.

The urine remained practically normal throughout the illness.

There were constantly six attending physicians and surgeons on this case. Three changes were made in attending surgeons and there were several consultants. One of the attending physicians made the statement to the family in my presence that the boy could not possibly recover. This was about the middle of his illness, at the time that the suppurative arthritis of the hip developed. This naturally discouraged the parents, who announced that they wished nothing more done except to keep the boy as comfortable as possible. Some of us, however, felt that we had a fighting chance and persuaded the family to let us carry on. It was a grand fight to win.

I do not wish to overemphasize the part that transfusions played in this boy's recovery, but it was perfectly apparent to all that without this supportive measure he could not have survived.

DR. J. G. DWYER, New York: Transfusions constitute, in my opinion, the greatest advance we have made in otolaryngology in recent years.

I wish to congratulate Dr. Shea from two standpoints: one of them, on giving us a good working rule-of-thumb method of diagnosing our cases from the transfusion standpoint. I want to take this occasion of congrat-

ulating him personally on the work he has been doing these recent years. I have been following this year after year. He was with me some years ago when we started out. I think it is perfectly wonderful.

I am going to approach the question from two or three standpoints, that neither Dr. Shea nor Dr. Stetson have touched upon.

First, I would like to emphasize the early use of blood transfusion. There is a fear among the laity that the minute you mention the transfusion process that means that the patient is going to die. We ought to get that fear out of people's minds and impress upon them, as Dr. Shea has said, that it is really one of the best tonics that we can use. It is far better to prepare the patient first rather than deal with complications afterwards.

At the Manhattan Eye and Ear Infirmary, we have been using repeated small transfusions rather than having large transfusions.

I wish to emphasize a point that struck me all of these years, that there is a definite relationship in my mind between the weights of the patients. We get far better results all the way through with children than we do with adults. I am coming to that afterwards.

Another question that has not been touched upon very much is the question of immunization. I thoroughly realize when we make a diagnosis of hemolytic streptococcus we know nothing at all about the strains of the streptococci. In some of my cases I have taken the blood of patients and tested that out against the blood serum of the donor and found an immunized donor had been immunized against that strain and we got perfectly startling cases where in the same case we had given blood transfusions of ordinary blood.

Emphasizing the point that I made about the relationship of the weight of the patient, that is, the volume of blood from the donor into a person, we had within the last ten years two of the worst complications that we could possibly have in our work. A child was operated on for double mastoid. We had no diagnosis whatever on which side we should go in. We decided to transfuse them and the improvement was so marked with the blood transfusion alone that we never had to tie off the jugular.

In addition to matching up the blood, we ought to test out the culture that we obtain from the patient against the various donors' blood. We can do that in New York where we have a lot of donors on hand.

Then I want to emphasize before closing that we ought to try to remove from the patient's friends or relatives the fear that a transfusion is the final thing. I wish to take this opportunity of thanking Dr. Shea and Dr. Stetson for placing a question like this in the literature so that when we are up against it we can use it as a guide and rule-of-thumb.

DR. J. I. DOWLING, Albany, N. Y.: It is from the clinical standpoint that I wish to discuss this paper.

During my rather long experience I can recall seven cases in which blood transfusion has undoubtedly saved the lives of patients. Some of those have not been mentioned here today as necessary for the purpose.

One case that I had was a rather interesting one. I stepped into Southampton Hospital after having come in from a game of golf. I found a patient with bright red blood gushing from the mouth. I took him to

the emergency room, opened his mouth and packed the cavity which extended under the tongue with cotton saturated with pure dioxygen. Under the direction of Dr. Fenton Taylor there was a ligation operation performed and blood transfusion given. The case was one of Vincent's angina. There was an erosion in the lingual artery and had prompt measures not been taken that man would have lost his life. I think the blood transfusion and the tying of the carotid had a definitely valuable effect.

In regard to tonsils, a great many men, and particularly the younger men, if you speak of hemorrhage in tonsils will say, "I have done so many hundred or so many thousand. I never had a hemorrhage."

I will say to them that if they will operate long enough and will take the cases as they come, the severe cases, the old cases of quinsy, streptococcic infection, and they will operate those cases as they do the children and they will, sooner or later, come to a case where they have a severe hemorrhage. For that they need not be ashamed. One of our very prominent clergymen had his tonsils out and after taking out the first — and his blood clotting time was four minutes — he had such a severe hemorrhage that I had to insert a pad and sew it in place and then give a blood transfusion before going to the next. Later on the other tonsil was taken out, after his throat had been cleaned up.

In another case, which was probably inexcusable, I operated a case of old quinsy, and I thought I had it controlled. I asked the advice of others who were present and at least three surgeons said, "You don't need to put any pads there." Although I had qualms, I did not put any between the pillars of the throat. The result was that I had a severe postoperative hemorrhage and I had a great deal of difficulty in controlling it. But, blood transfusion in that case saved her life.

Dr. A. A. Eggston, New York: It has been my opportunity and pleasure to be associated with an otologist and otolaryngologist and pathologist for the last sixteen years at Manhattan and in this position have been more or less asked, and have been interested in doing, blood transfusions on certain cases.

In this period of time, I have seen a tremendous change in the opinion of the men approaching transfusion, and I myself have changed. I have become enthusiastic about the effect and the benefit that these patients receive, particularly the septic cases. Hemorrhage, of course, is itself a simple thing and of course you just add what nature has lost.

There has been some argument about whether transfusions help in infections. I do not think anyone can have much experience with these septic cases, particularly with positive blood cultures, without being convinced that a lot of lives are saved by the transfusion of blood in these cases.

I certainly believe with Dr. Shea that blood transfusion should not be allocated to internes and should be done by men of experience who know how to determine the size of the dosage. Sometimes we give larger doses, sometimes we give smaller ones. It should also be said that hematologists should more or less decide when the transfusions should be repeated.

I am glad that Dr. Shea brought out the fact that blood counts are of no value in deciding when the blood transfusion should be repeated.

You do not give transfusions for hemoglobin and red cells in an infection; you give it by the supply of protein or immune bodies you are fortunate to have found.

The protein is a very important factor, particularly in sepsis. A lot of leukocytes have been destroyed and the pus throws off a lot of protein. I think one of the beneficial effects of giving blood is the addition of the proteins.

There is the question of technique. While blood has helped in cases of hemorrhage, I do not think that it has the beneficial effect of fighting infection processes.

Since I have been at Manhattan I am quite confident in saying that our deaths from septic thrombosis have been greatly reduced as a result of giving proper transfusions at the proper time and in the proper places and in cooperation with the surgeons.

Our technique there is, in cases where there is a positive transfusion, to transfuse before the operation and to transfuse as necessary after the operation.

I certainly want to voice my appreciation for Dr. Stetson's discussion of this paper because he certainly has had a tremendous experience with it. I think it is a timely subject to bring before this body.

A MODIFICATION OF THE BALLANCE-DUEL TECHNIQUE IN THE TREATMENT OF FACIAL PARALYSIS*

JOSEPH A. SULLIVAN, M.B.

TORONTO

IT WOULD be presumptuous to proceed with this presentation without voicing what I feel is in the minds of all otologists, namely, the irreparable loss that we as a society have sustained in the unfortunate deaths of Sir Charles Ballance and Dr. Arthur Duel. The history of facial paralysis is imperishably linked with their names. It was my good fortune to have been associated in the rôle of pupil with the late Sir Charles and to be aided by his distinguished coworker and colleague, Dr. Duel.

In the gradual evolution of truth and the end of each stage of medical cycles, it would seem that a great man or men appear who focus in their speech, writings or actions the best thought of the preceding era; or who are the messengers of a new knowledge, the heralds of the dawn of a wider insight into life or scientific progress. Some names mark epochs in the advancement of knowledge, Hippocrates, Celsus and Galen represent the highest development of clinical medicine and surgery unsupported by accurate anatomical and pathological learning. Morgagni, anatomist and pathologist, bridges the gap between the famous anatomists of the middle ages and the great pathologists on the nineteenth century, who came just when the knowledge of morbid anatomy seemed "to have reached its last finish."

Was it not Paré, in the preface to his great work on surgery in 1582, who said in effect that excepting unimportant details, nothing more was to be added to the science and art of surgery? But "there is no last finish" in the path of medical progress. In the fullness of time, the experimental method of scientific research has invariably culminated in the establishment of definite clinical results. Nowhere has that procedure, in the entire annals of med-

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icine, been more conclusively demonstrated eventuating in a successful outcome than in the work of Ballance and Duel. Their advent has opened up new fields of pursuit and practice and has given promise of a wider hope and further triumphs to the student of surgery. Therefore, prompted with that thought and fortunately situated so as to partake of their scientific endeavour and fullness of knowledge, the following is presented.

The accepted surgical procedures to date for the cure of facial palsy are:

1. *Anastomosis operations* — anastomosing the peripheral end of the facial with the central end of another nerve.
2. *Plastic operations* — this abandons the paralyzed muscles and seeks to mitigate the deformity by muscular substitution or purely mechanical means. This procedure is feasible and only feasible in those cases in which no muscular tissue is left in the face.
3. *Decompression of the facial nerve* — subdivided as follows:
   a. simple removal of a depressed fracture
   b. end to end apposition with or without suture
   c. interposition of a homogeneous graft.

The recent research work and practical clinical experience has conclusively proven and shown that the method of restoring lost facial functions by union of the facial nerve with one of the adjacent nerves in the neck is deprecated, and, to my mind, in place of this method the employment of an autoplastic graft is advised to bridge the gap from the proximal to the distal segments caused by injury or disease.

In experimental work that I have carried out and from my actual clinical experiences a phenomenon occurred which has interested me greatly. Ballance, in speaking before the Royal College of Surgeons in England in July, 1934, stated that in their experiments on animals, spontaneous contractions of the muscles of the face were observed.

If such an animal after a nerve grafting operation is lightly anæsthetized, the twitch or spasm becomes more pronounced, that is, anæsthesia cuts out the control of the neopallium. To quote Ramsey Hunt, "The spasmodic manifestations in the kinetic sphere occur as the result of irritation, or as release phenomena in subordinate motor systems due to loss of high inhibitory control." Hunt designates the cortex as the neokinetic level having

to do with isolated movements, the subcortical region as the paleokinetic level having to do with associated movements, and the segmental nervous system or medulla spinal cord region concerned with reflex movements.

If this animal has its cerebrum removed by section of the crura cerebri, the carotid arteries being clamped to avoid hemorrhage, the spasms of the facial muscles become still more exaggerated: the animal continues to breathe and live on its brain stem. This experiment demonstrates that "the paleo-encephalon, represented by the corpus striatum and optic thalamus which constitute the higher coordinating sensory and motor mechanisms of the lower forms of life, has, like the neo-encephalon, some control over the facial spasms and therefore the removal of the paleo-encephalon is associated with wider and more marked muscular spasm.

The segmental nervous system, as described, which contains the reflex systems of the neuro axis, and is the archaic representation in man of the nervous reactions of the lowest forms of life, according to Ballance is the explanatory site of the spontaneous muscular contractions. It is known that section of the trunk of the facial nerve is associated with regressive changes in the pyramid cells of the facial area of the opposite Rolandic cortex, and now Ballance has shown there are similar regressive changes in the cells of the facial nucleus in the medulla (Figs. 1 and 2).

These latter changes are the essential cause of the muscular spasms of the face which have been observed in experimental animals. They are really local epileptic phenomena which are caused by disorder of cell functions. They are never the result of obstruction to the passage of nerve impulses travelling along nerve fibres, but rather a disorder of the cells of the facial nucleus surrounded by tissue and vascular changes. He stipulated that neither Dr. Duel nor he had observed, after operations on patients for paralysis of the face, any spasm of the muscles. To postulate my conclusions, it is necessary to state the following: In experimental work on animals the facial palsy is recent. It dates from the time of operation. With the section of the facial nerve the cells of the facial area of the opposite Rolandic cortex and the cells of the facial nucleus in the medulla commence to degenerate. Cajal clearly defines and shows these important changes occurring in nerve centres after nerve sections. The truth of the matter is that the cell of origin or trophic centre whose

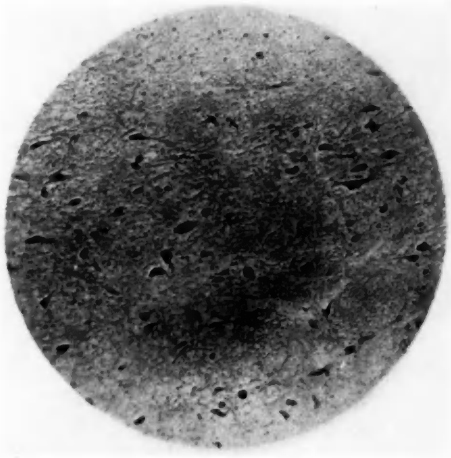


FIG. 1 — (Sullivan) — The cells of the normal nucleus in the medulla pontine region of a horse. *(Courtesy of Sir Charles Ballance).*

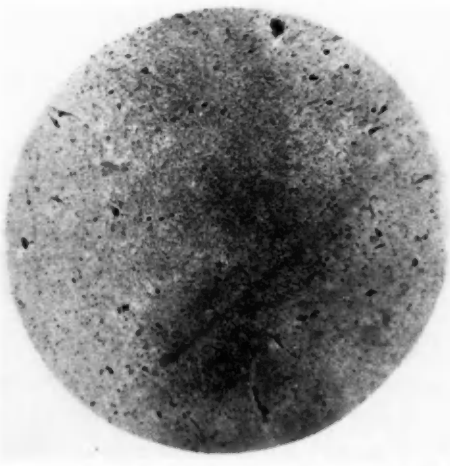


FIG. 2 — (Sullivan) — The corresponding area on the opposite side, many cells have disappeared and those remaining are undergoing regressive changes as a result of facial paralysis of three months' duration. *(Courtesy of Sir Charles Ballance).*

axon is mutilated or even functionally isolated shows morbid changes and may even die. Paradoxical as it may appear and contrary to the usual course of science, "error is modern, while truth is ancient." The degeneration process ceases at any rate in these cells if the operation is followed by the passing of nutritional impulses within a reasonable time to the paralyzed muscles. The cells slowly regenerate. During all the time of degeneration and of regeneration the cells are in a state of physiologic disorder. They are in unstable equilibrium. This is the explanation of the spasm symptom. Now compare the condition of the facial medullary nucleus of a patient with facial palsy with that of the cells of the facial nucleus of an experimental animal. In man, the operation for the cure of facial palsy is often delayed for many months or even years in the vain hope of recovery of the palsy without operation. The cells of the facial medullary nucleus are atrophied. Tissue and vascular activity are in a state of stable equilibrium. There are no degenerating processes going on and no associated vascular changes proceeding. Ballance suggests this as an ample explanation of the fact that in man facial spasm following a remedial operation is not observed.

The restoration of the movements of the paralyzed face, and in part with self-education, return of emotional response without associated movements is an incalculably valuable rendition of these men to the neuro-surgical world. However, gentlemen, "facial spasm can and does occur in the human"; just as important as it was to eliminate the occurrence of associated movements, nearly equally as important is it to eliminate the occurrence of facial spasm in order to attain a result or results approximating a standard of perfection as it is humanly possible to do. More advantageous than animals to evaluate results so as to be conclusive are human patients. To clarify and definitely substantiate this assertion the following cases are presented:

Case No. 1 — R. B., Age 22:

1. Complete facial paralysis, three months' duration, following mastoid operation July, 1933.
2. Nerve graft preparation November 6, 1933.
3. Radical mastoid operation and decompression December 1, 1933.
4. Nine millimeter graft placed in the pyramidal segment.

CASE NO. 2 — B. K., Age 20:

1. Practically a complete facial paralysis, fourteen years' duration following mastoid operations in 1919 and 1920, during which time galvanic stimulation and massage was applied to the face.
2. Nerve graft preparation January 20, 1934.
3. Secondary radical mastoid operation and decompression February 7, 1934.
4. Eleven millimeter graft placed in the gap occupying part of tympanic and mastoid segment.

It will be observed from the two cases presented that in the first spontaneous contractions of the muscles of the face occurred when a remedial operation was performed three months after the paralysis was produced. In the latter case a paralysis of fourteen years' duration was present when, after a remedial operation was performed, an almost complete recovery occurred, without facial spasm. The difference, from what was apparent in the foregoing part of the paper, seems obvious; nevertheless, I can assure you it was not; when confronted with this problem, only after long experimentation, trial and error, was a concrete solution deduced. In the first case, there is physiologic disorder of the cells of the facial medullary nucleus and in the latter case these cells are atrophied. "That is the difference."

The question of how the brain cortex reassumes control of the facial muscles is best understood by the study of other cases in which conditions present are somewhat comparable to facial palsy after a remedial operation has been done. To quote Spiller, "When the speech centres of the left side of the brain are destroyed in early life the power of speech usually returns. It is assumed that the dormant centres on the right side of the brain acquire the function that would otherwise belong to the centres of the left cerebral cortex. Here is an illustration of the education of a new centre in the brain for the performance of a function after the usual centre is destroyed." It is possible that such a case is exactly comparable to one of facial palsy for which a remedial operation has been performed, and that the cells in the facial area of the opposite Rolandic cortex atrophy and become no longer of any service in the case of a facial palsy of about six months' standing. The expression of the emotions is the best test as to whether the operation has been followed by the harmonious working of the two sides of the face, this depends to a great degree on the perseverance of the individual to re-

educate his or her facial muscles. The allotted time for the reading of this paper does not permit of entering into detail concerning the neurological concept of motility. It is a complex problem and I refer you to the writings of Ramsay Hunt on the "Dual Nature of the Efferent Nervous System" for elucidation of this subject and the interpretation he places on these various levels in their relation to motility. Sufficient is it for me to say that in the discussion of Dr. Duel's paper before the American Otological Society, May, 1933, Hunt remarks that it will be interesting to watch these experimental animals and also human subjects to see if after a clear cut operation neo-kinesis will return. Gentlemen, not only does neo-kinesis return, but is there not evidence that we have the centre of the opposite cortex acquiring the function of the usual centre? I believe there is, as is shown in Case No. 2.

Briefly, the explanation Hunt puts forth is, looking back over the phylogenetic development in animal life, that we can trace this problem of motility in the increasing complexity of the central nervous system. The lowest forms show reflexes and these are represented in the segmental nervous system or archeo-kinetic system. Further on it becomes necessary to harmonize the reflexes and the paleo-kinetic or extra pyramidal system comes into development. The centres for this system are at the thalamostriate level. The thalamus represents the sensory system and the striate level the motile system. The spinal cord represents the system which covers automatic associated movements, such as running, walking, advance, orientation, movements of expression of the face, etc. The next development is the pyramidal tract or corticospinal system. The impulses of the Rolandic region of the cortex pass down through the capsule, through the whole length of the brain stem and spinal cord to the anterior horn cells. This is the neo-kinetic system and has to do with control of isolated movements. The peripheral nerves have their physiological representatives in the paleo-kinetic and the neo-kinetic systems. The lower type system is less vulnerable and restoration takes place more rapidly so that the nerves reach their destination more quickly, whereas the neo-kinetic fibres are more highly specialized and would therefore regenerate much more slowly.

It is only fitting and proper that I should state that Dr. Duel saw these cases in Toronto in May, 1935, and very kindly offered to honour me by discussing this phenomena when the subject was presented.

In regard to the question of nerve grafts, I wish to state that their use in surgery is by no means an entirely new idea. It has long been known that the function of a divided nerve might be restored by the introduction of a graft into the gap; it has been employed in the soft parts for many years. Success, moreover, has followed upon the use of grafts in other regions much longer than any which could ever possibly be required in the repair of an injured facial nerve. The reason that it was not long before attempted at this site was clearly because the facial nerve, in its course through the temporal bone, is enclosed in a bony tube of ivory-like density. I am quite cognizant of the fact that in the development of peripheral nerve surgery of all methods devised, that of nerve grafting holds the most pessimistic outlook among many surgeons. Although there has been abundant brilliant experimental evidence in support of this method, actual clinical and practical experience, so far, has not completely warranted its usage in these regions. Why? Dr. Duel's great contribution elucidates the possible answer which may be interpreted thus: "Whenever and wherever a nerve graft is required in surgery, a degenerated graft should be employed. The fresh nerve graft is abolished from surgical practice." Cajal was the first to foretell the practical value of the degenerated graft. He quotes "The ideal graft is the peripheral stump with bands of Büngner newly taken from an operated animal eight to fifteen days after operation." (The bands of Büngner are the phase of longitudinal striation which appears in the distal segment during the degeneration process). The newly-formed fibres travel through the empty sheaths with extraordinary speed, deviations and retrogressions being much diminished. It would seem that Cajal was led to recommend the use of degenerated grafts by the experimental work of Tello who implanted portions of the sciatic nerve into the cerebral substance. Fresh grafts were absorbed while degenerated grafts attracted the nerve fibres of the brain. These penetrated the open ends of the prepared grafts. Tello states "that the empty channels of the degenerated grafts are especially rich in neurotropic substances, whereas in the normal nerve grafted directly, though not entirely absent, they are liberated very tardily."

In respect to the question of nerve degeneration and regeneration, there have developed two schools whose disciples agreed as to *what* took place, but not as to *how* it took place. It is an obvious and well known fact that a divided nerve suffers a trau-

matic degeneration for a short distance in the cut end of its proximal segment and completely throughout its distal segment, namely, wallerian degeneration in the latter has occurred. If a nerve when divided is immediately re-united there follows a period of hesitation before any recovery begins to be manifest, because this wallerian degeneration in the distal segment must take place and the paths be at least partially cleared before the new axons from the proximal segment can travel through. Cajal has shown that a grave cause of retardation and perturbation in the growth of sprouts is the fortuitous presence in the scar of adipose lobules or blood clots that have not been resorbed, with the greatest obstacle being foreign bodies such as the threads of a suture. In peripheral regeneration it seems necessary to postulate the existence of alluring or attracting substances, both in the peripheral stumps and in the terminal structures. These substances have the faculty of orienting or facilitating the growth of the axons in determined paths. It is a fact of our clinical observation corroborated by accurate histo-pathological findings that the greatest neuro-trophic influence of the peripheral stump coincides not with the phase of the bands of Büngner, but precisely at a time when most of the myelin has been resorbed.

I am indebted to Dr. Eric Linell, professor of neuro-pathology at the Banting Institute, University of Toronto, for the cutting of these sections of nerve tissue and his interpretation.

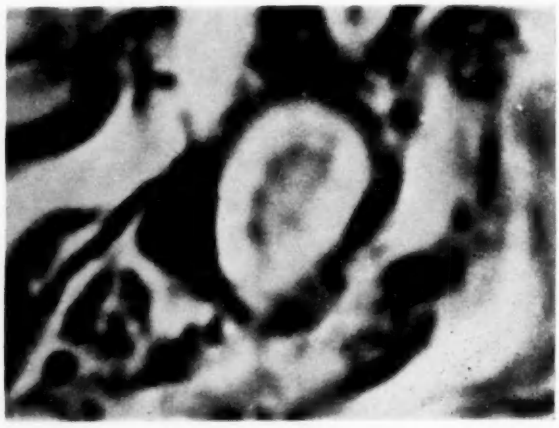


FIG. 3 — (Sullivan) — A high power photomicrograph of a single normal medullated nerve fibre (prepared by Dr. W. T. Grant). This shows from without inwards the neurilemmal sheath with its oval dark-staining nucleus to the left of the fibre. The clear space within this sheath is the myelin sheath, within which again the granular dots represent the cut ends of neurofibrils.

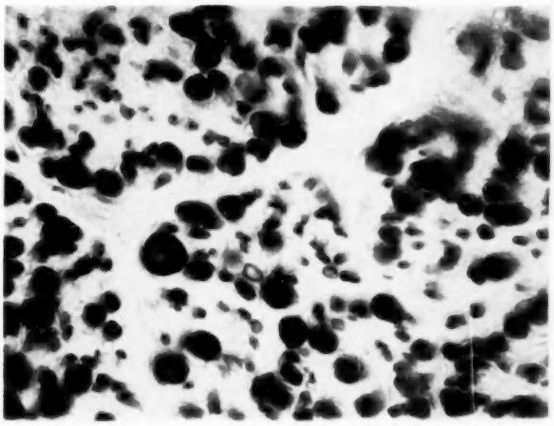


FIG. 4 — (Sullivan) — A high power photomicrograph of the distal end of the lateral femoral cutaneous nerve 9 days after section, stained by Weigert. This shows the changes of early wallerian degeneration in the nerve fibres by the fact that, in the majority of the large fibres, the myelin sheath is not clearly cut and the lipoid is invading the central area of the nerve fibre, normally occupied by neurofibrils. Slightly to the right of the centre of the field a fibre can be seen still showing evidence of a central light area. Considerable numbers of small fibres are present with well-defined, thin myelin sheaths.

FIG. 5 — (Sullivan) — Distal segment of sciatic nerve 9 days after severence, in young rabbit, showing bands of Büngner (Hæmatoxylin and Eosin preparation × 250).

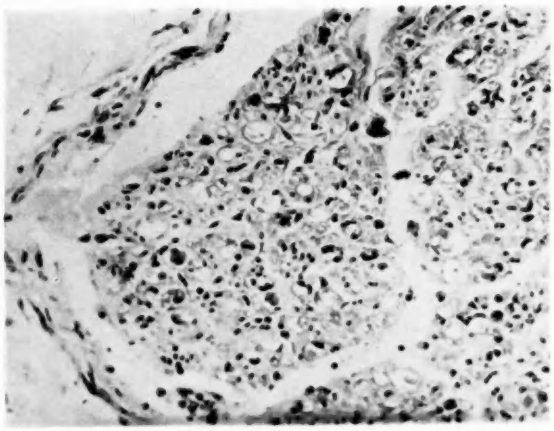


FIG. 6 — (Sullivan) — A transverse section of the distal segment of the lateral femoral cutaneous nerve, 27 days after section, stained with Cresyl Violet to show the open, distended neurilemmal tubes of the degenerating nerve. Small fragments of degenerate myelin can still be seen within some of these tubes.

FIG. 7 — (Sullivan) — A longitudinal section of the distal segment of the lateral femoral cutaneous nerve, 27 days after section, impregnated by Bielschowsky. This shows a few broken up fragments of degenerate neurofibrils. (Dr. M. Thompson).

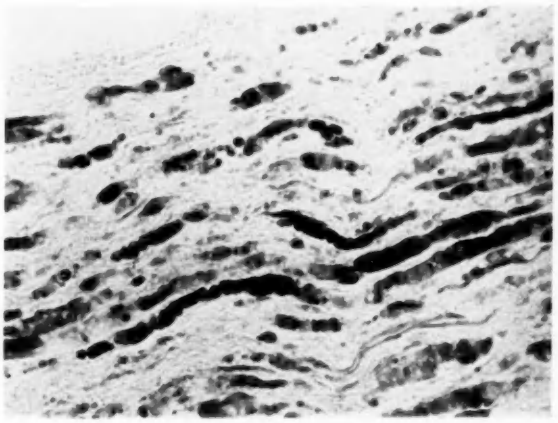


FIG. 8 — (Sullivan) — Longitudinal section of distal segment of lateral femoral cutaneous nerve, 27 days after section, stained by Weigert. This shows severe wallerian degeneration with the breaking up of the myelin of the medullary sheaths into small globules. The continuous medullary sheaths, which are still present, show marked irregularity of their contour, due to the contained irregularly-sized globules of degenerating myelin.

The clinical findings of this process are exemplified in Cases 3 and 4:

CASE No. 3 — J. C., Age 25:

1. Complete facial paralysis following a radical mastoid operation.
2. Decompression within seventy-two hours of injury, July 22, 1935.
3. Nerve was found with spicule jammed into its substance, producing complete division, spicule removed, sheath slit, and ends approximated.

CASE No. 4 — W. G., Age 40:

1. Complete facial paralysis following radical mastoid January 26, 1935.
2. Decompression within seventy-two hours of injury.
3. Nerve completely divided, bony spicule removed, sheath slit, the ends were not apposed.
4. Three weeks after injury the nerve ends on being freshened were approximated, no suture.

CASE No. 5 — W. T., Age 22:

1. Practically a complete facial paralysis, five years' duration, following a so-called simple mastoid operation December, 1930.

2. Nerve graft preparation February 7, 1935.
3. Radical mastoid operation February 27, 1935.
4. A partial previous splicing of nerve had occurred just distal to horizontal semicircular canal — two ends freshened and approximated; a bony spicule in tympanic segment removed, no graft.

This case is presented to show that excision of the damaged portion of the nerve and use of a nerve graft are not always necessary. After a compression of five years' standing, yet with a decompression only and proper apposition, the recovery is more than to be expected. The histo-pathological findings in a nerve which has been moderately compressed shows a superficial region rapidly necrosed and a deep region where the axons survive in a phase of formative excitation or turgidity, by that I mean a state of amœboid division of the axon, that is a rejuvenescence of the axon.

Case No. 6 — Mrs. D. W., Age 26:

This case is unique in the fact that the paralysis occurred concomitant with fracture of the skull, requiring an unlocking of the nerve in the petrous pyramid beyond the geniculate ganglion and using a twelve millimeter graft.

1. Head injury, July 30, 1934.
2. Nerve graft preperation, October 10, 1935.
3. Decompression following radical mastoid operation, October 30, 1935.

Conclusion

(1) Facial paralysis immediately following a mastoid operation warrants exploration.

(2) Facial paralysis of six months and less which would require a graft to replace the gap caused by removal of the injured nerve segment should not be undertaken in that interval. Primarily, to eliminate the occurrence of spontaneous contractions of facial muscles, by allowing the facial medullary nucleus to atrophy, such a procedure resulting in a much more perfect functional recovery. "On this earth of ours, what great good has ever been or can be gained except by sacrifice? It is in every era and in every zone the law of life."

(3) A traumatized nerve with complete section and such that its ends can be joined, it is suggested that one wait three weeks before approximating to eliminate the band of scar tissue which invariably forms between the two segments, thus allowing wallerian degeneration to occur, as it always does, in the distal segment and producing thereby an ultimate result of recovery as near perfection as is surgically possible. Tello's work on degenerated grafts, Cajal's researches on the growth of sprouts through the scar, our observations on the most receptive stage of the degenerated graft with the definite clinical findings and improved results which followed this procedure in the surgery of the facial nerve corroborate and add authority for the justification of this statement.

(4) Finally, there is every reason to believe that a direct line of repair can be undertaken in facial paralysis following fracture of the skull, and it need not be relegated to the surgeon's old love, so-called nerve anastomosis.

* * *

I am signally honoured in having Dr. Duel's lifelong friend and associate, Dr. John R. Page, open this discussion.

I owe a deep debt of gratitude to Dr. J. X. Robert, chief of the otolaryngological service, St. Michael's Hospital, Toronto, for permitting me to carry out the operative procedure on these patients in his department. I am especially indebted to Dr. Eric Linell, professor of neuro-pathology of the University of Toronto, for his sectioning and interpretation of the nerve tissue specimens, coupled with his valued counsel and aid in their clinical application. I wish to express my thanks to Dr. W. E. Gallie, professor of surgery, and Dr. Perry Goldsmith, professor of otolaryngology, in the University of Toronto, for affording me the opportunity to carry out the experimental work in their laboratories of the Banting Institute, Toronto.

(Cases described and illustrated by moving pictures.)

DISCUSSION

DR. JOHN R. PAGE, New York: I was interested to read Dr. Sullivan's paper, which he very kindly sent me before this meeting, and it is gratifying to be invited to open the discussion of it even though the honor is not merited and I feel that the invitation is due rather to my friendship and long association with Dr. Duel than to any knowledge of the subject I

might have. I am glad that Dr. Tickle, who is now in charge of the facial palsy clinic that has been established as a memorial to Dr. Duel at the Manhattan Eye, Ear and Throat Hospital, is here to speak with greater authority than I because of his experience and close assocation with Dr. Duel and Sir Charles Ballance from the beginning of their work.

I agree with Dr. Sullivan's first conclusion in the summing up of his paper, and I feel sure that Dr. Duel, too, would agree that "Facial paralysis immediately following a mastoid operation warrants exploration." It is what he preached, and is what I am sure we all agree to for the obvious reason that one should not shut his eyes in the face of a bad situation that might be relieved by seeing what the cause of it is. Then, too, as Dr. Tickle says in a paper presented jointly by Dr. Duel and himself (at the New York Academy of Medicine, published in the Annals of Otology, Rhinology and Laryngology last March): "If an injury to the facial nerve has caused paralysis of the facial muscles an immediate investigation has the advantage that the segment of the facial nerve distal to the injury retains response to Faradic stimulation for from forty-eight to seventy-two hours. No matter where the injury is the advantage of being able to identify the distal segment by Faradic stimulation is of great advantage." Dr. Sullivan's experiments upon which he bases his second conclusion that cases of facial paralysis of six months and less which would require a graft to replace the gap caused by removal of the injured nerve segment should not be operated upon in that interval in order that the facial medullary nucleus be allowed time to atrophy and so prevent the occurrence of spontaneous contractions of the facial nucleus are contrary to Dr. Duel's experience and to the investigations carried on at his laboratory by Dr. E. P. Fowler, Jr., of New York, and Drs. Tower and Howe of Dr. Lewis H. Weed's staff from Johns Hopkins. These investigations were referred to by Dr. Duel in the year book of the Carnegie Institution of Washington 1934-35, under the title of "A study of Spasm or Tic occurring in Facial Muscles Recovering from Facial Palsy." Additional interest was aroused in this investigation by the difference of opinion that was held by Sir Charles Ballance and Dr. Duel at that time. Dr. Duel says an animated correspondence took place between them, he contending that the defect must be peripheral and Sir Charles that it must be central. I am sure that those who have not read this report will be interested to hear from Dr. Fowler, who was one of the investigators at that time. Suffice it to say in Dr. Duel's words "The final answer, as will be seen from a study of the long report, is that the tic occurring in recovering facial palsies in monkeys is due to a splitting of the axones in the peripheral branches. Once established, this tic shows no sign of regression."

In the one hundred and twenty or more patients that were operated on for facial palsy by Dr. Duel and Dr. Tickle, I have never heard from either of them that the occurrence of spasm was more prevalent among those operated on early than among those operated on late. The facts regarding this distressing phenomenon should be obtainable, and no doubt can be given us today by Dr. Tickle. It would be interesting to know what percentage of cases in this comparatively large series developed the spasm, and at what time after the paralysis those that did not were operated upon. It seems possible to me that Dr. Sullivan may have misread or misunder-

stood Sir Charles Ballance when he quotes him as saying that Dr. Duel never observed the development of any spasm of the facial muscles after operation on any of his patients, and that in man facial spasm following a remedial operation is not observed. Although I believe Sir Charles' personal operative experience in this work was limited to operations on animals it is not likely that in the active correspondence that took place between Dr. Duel and himself on the subject of tic that he would not have learned of Dr. Duel's later experience with it in man. There are others here who from experience and research know more about this subject than I do, and I hope time will be allowed for us to hear from Dr. Tickle and Dr. E. P. Fowler, Jr.

Dr. Sullivan is to be commended for his interest and zeal, and I will congratulate him when he proves right in his findings regarding the control of this distressing tic that has baffled all up to the present time.

Dr. Thomas G. Tickle, New York: I have listened to Dr. Sullivan's paper with great interest and I think that he is to be commended on his close observations in his cases with facial tic; but I am sorry I cannot substantiate his interpretations as to why this occurs. A large number of our cases that have been operated upon for facial paralysis of more than six months' duration have now developed a tic. Some of the cases that we have operated upon within 48 hours from the onset of the facial paralysis have not as yet developed one. The first case of Bell's palsy that was operated upon by the late Dr. Duel and myself was a young lady who had a facial paralysis of 10 years' duration. She now has a definite twitch. I would like to say that all of our nerve graft cases have an associated movement of practically all the muscles on that side of the face, showing that there is a jumbled distribution of the axones, or what is known as split axones, as shown by Dr. E. P. Fowler, Jr., of New York, and Drs. Tower and Howe of Dr. Lewis H. Weed's staff from Johns Hopkins.

Now as to the question of degenerating the nerve before using it to bridge in the gap of the destroyed facial nerve: The late Dr. Duel thought that this would shorten the time of recovery and possibly improve the end results. Theoretically this sounded plausible for when you cut a nerve, allowing it to remain in situ for from 14 to 21 days, it undergoes what is known as wallerian degeneration, thus leaving a set of empty tubules ready to receive the neurons from the proximal end, but, gentlemen, very few neurons go through the tubules, as was formerly thought, most of them go along the sheath on the outside to the distal end of the facial nerve.

This has been proven by Mr. L. H. Bentley at the experimental laboratory of the Royal College of Surgeons.

Experimenting on cats, both fresh and degenerated grafts of the same lengths were used, and then comparing the length of time before voluntary motion was restored. His findings being that motion was restored as quickly with fresh grafts as with degenerated ones. The cats were

then killed and the grafts examined histologically to determine why this occurred. The answer being that few neurons go through the empty tubules, as I mentioned above.

This also coincided with my clinical observations in over 100 cases operated upon by the late Dr. Duel and myself, that facial motion was restored as quickly with fresh grafts as when degenerated ones were used.

Dr. E. P. Fowler, Jr., New York: I would also like to congratulate Dr. Sullivan on his work with facial paralysis because he got such nice results. However, I cannot agree with him about his interpretation of facial tic.

If you noticed the first case that he showed on the screen you saw that concomitant with each tic or twitch of the face there was a blinking of the eye not only on the same side but more marked and more plainly seen on the other side.

The second case did not wink his eye. He had a very good poker face and therefore he did not have tic.

The answer is found in the later work of Dr. Duel, which I happen to be associated with, in bringing out that Sir Charles Ballance was quite wrong in his interpretation of the tic; although he was the first one to observe the tic in the animals, yet his conclusions and interpretations have been shown to be incorrect.

It has been known for a great many years that if you cut a motor nerve the regenerating nerves, the axones of regenerating nerve, split at the point where the nerve was cut. What happens is that a single axone grows down into a neuroma, is all jumbled up, and certain parts of the axone shoot neurites out to one part, and certain parts shoot axones to the other parts. The result is that the eye on one side can be ennervated by exactly the same axone as the lip, for example. If the same axone ennervates both eye and lip, the same cell in the medulla is also ennervating both eye and lip and therefore the patient tries to close his eyes and when he does he is going to move his lip.

That gives an associated movement which is always present in the case of regeneration with a cut nerve. It is not always present in regeneration with an uncut nerve. It is always present, to some extent, in every single animal and every single patient which I have seen. It also gives rise to the so-called crocodile tear phenomenon. There are sympathetic fibers in the face which ennervate the lacrimal and salivary glands. The result is that certain of these patients if they have very active lacrimal or salivary secretions will, after the tear, develop tearing of the eye every time they eat. Great tears roll down one side of the face. It is a very annoying thing when it happens.

As for the findings of Sir Charles Ballance, Sir Charles was good enough to send to me his sections of the cortex, but I have been unable to substantiate his findings in the 40 monkeys that he left here after he went away. We took out the Rolandic cortex of the facial area on both sides. I examined them in the laboratory. I have been unable to find any changes in the Nissl bodies which were not an artefact. I could not find very much difference in the findings of Sir Charles of that type. I was

very much upset about that and showed them to the neuropathologist in the city who pointed out that the little squares in the photograph showed that Sir Charles had taken the degenerated side. The pyknotic cells which he showed on the degenerated side were all on the edges of the section and the normal ones he showed in his book were all in the middle of the section. It is a very common thing, he says, for the edges of a piece of brain tissue to show pyknotic cells. Therefore, that seems to take the ground out from under Sir Charles' feet on that particular finding.

Furthermore, Dr. Sullivan pointed out, a few moments ago, that Cajal had shown changes in the parietal cells of the medulla in cases where peripheral nerves had been cut. I think he will find that most of the literature shows that the only time there are changes high in the brain stem in cases where peripheral motor nerves have been cut and are present is when the nerve is cut very close to the spinal cord. If you cut it anywhere peripherally there are no changes centrally.

DR. JOSEPH A. SULLIVAN, closing: I wish to thank Dr. Page, Dr. Tickle and Dr. Fowler for their discussion of this paper.

If you cut a nerve coming off the spinal cord close, before it gives off any collateral branchings, you get degeneration of that nucleus. Suppose we have a section of the facial nerve which is distal to the stapedial branch, then we may not get a change in the facial medullary nucleus. The neurotropic centre has to keep up shooting impulses through. It has work to do. It might depend on the site of the lesion whether we have degeneration of the facial medullary nucleus or not.

In closing my discussion I would like to use the words of the immortal Carlyle, "The craftsman there; the smith with that metal of his, with these tools, with these cunning methods, how little of all he does is properly his work. All past inventive men work there with him as indeed with all of us in all things."

A NEW METHOD OF RADIUM APPLICATION IN CANCER OF THE BRONCHUS*

JOEL J. PRESSMAN, M.D., *and*
CLYDE K. EMERY, M.D.

LOS ANGELES

THE PRESENT-DAY treatment of carcinoma of the tracheobronchial tree consists essentially of four therapeutic measures — surgery, surgical bronchscopic diathermy, exposure to the X-ray and the local application of radium. None of these measures are adequate and very few cases of actual cures of this lesion have been reported.

It is not my intention to extol or decry any of these methods. My sole purpose is to present a new method of radium application. My excuse for so doing is the belief that radium has never had a fair trial because previous methods of application have been haphazard with inadequate dosage, irregular distribution, and failure to irradiate the surrounding tissues.

It is not within the scope of this presentation to enter into the anatomy, histology or pathology of these lesions. It suffices to remind you in order that we may understand each other's terminology that the tracheobronchial tree consists of the trachea dividing into its two main branches — the right and left main stem bronchi. The carcinomata which develop in these structures are either adenocarcinomata, arising from the numerous mucous glands which are distributed widely throughout the sub-mucosa, or squamous cell, arising from the lining epithelium which is stratified and columnar.

For purposes of future reference, I call your attention to the fact that this tumor, as well as the adenomatous variety, is medullary in type, being composed chiefly of epithelial elements and remarkably free from dense connective tissue. This is a very favorable factor in so far as radio sensitivity of these lesions is concerned.

* From the Bronchoscopic Clinic and Department of Radium Therapy of the Cedars of Lebanon Hospital, Los Angeles, California.

FIG. 1 — (Pressman and Emery) — The trachea dividing into its right and left main stem branches.

Early in their development, these tumors, as seen in Figure 2, are usually small, cauliflower-like growths involving either the trachea or one or the other of the main stem bronchi, and it is of tumors of this stage of development, size and position of which I speak.

Concerning the therapeutic possibilities of treating such tumors: I cannot enter into the controversy concerning the use of X-ray therapy, or diathermy. Experience would indicate that these are, to say the least, inadequate.

Surgery, in certain selected cases, is probably the definite method of choice, but in most cases, since the early tumors involve the upper portions of the bronchus and trachea, successful surgery is technically impossible. The site of these tumors which ordinarily makes surgery impossible, is, fortunately, the chief factor favoring the application of adequate doses of radium.

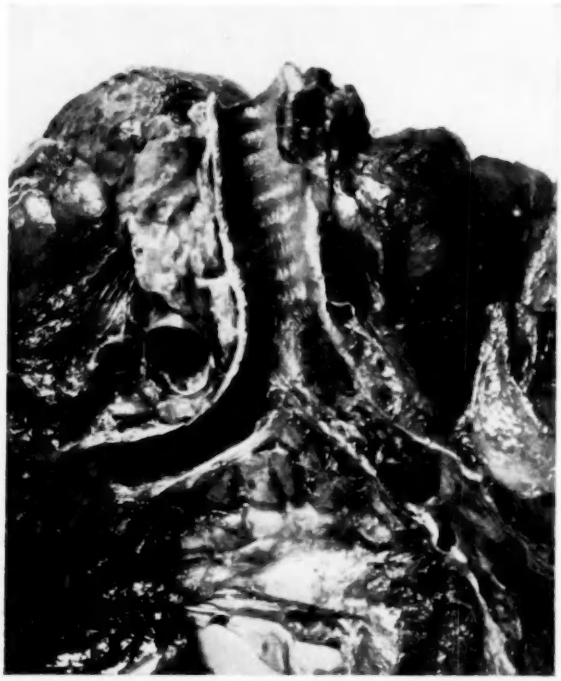


FIG. 2 — (Pressman and Emery) — Early carcinoma; right main stem bronchus viewed from behind.

There appears upon Figure 3 a white circle enclosing the lower portion of the trachea and the upper segment of both main stem bronchi. It is within the enclosure of this circle that the majority of tracheobronchial malignant neoplasms find their site of origin. This site is particularly favorable for radium therapy, because, as may readily be observed, there exists in most instances a sufficient segment of bronchus below the lesion, and a sufficient length of trachea above it to permit radiation beyond the limits of visible tumor. Sub-mucosal extension, within the lymphatics, or in the epithelium itself, can therefore be brought to fall within the zone of adequate radiation. This principle of establishing an effective field of radium activity above, below and to each side of the tumor, I believe to be of the utmost importance. No previous method of radium application has been able to accomplish it.

In overcoming obstacles of adequate radium application to tumors of the bronchus lies the sole value of this method which I am about to propose. Its limit of value, of course, is the theo-

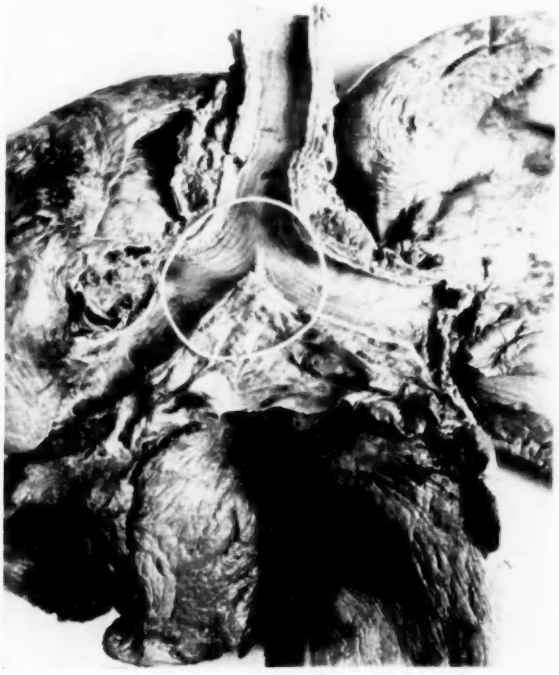


FIG. 3 — (Pressman and Emery) — The site of origin of most tracheobronchial tumors is located within the white circle.

retical limit of value of radium emanations in the treatment of cancer elsewhere in the body.

The method, in brief, consists of filling long, hollow tubes with ordinary radium needles containing the salt and implanting these tubes so that they fill the entire length of the main stem bronchus and portions of the trachea. These tubes are designed to fit the needs of individual cases and a great number of varieties are possible. By this means the entire bronchus and lower segment of the trachea is exposed to whatever dosage of radium is deemed necessary. This, therefore, includes the tumor and healthy tissue above, below and around it. This radium is in situ for as long a period of time as is deemed adequate.

The longer tubes, as shown in Figure 4, are 10 centimeters, or 4 inches, in length and from 6 to 9 millimeters in diameter. A determination of the size and type tube necessary is made at

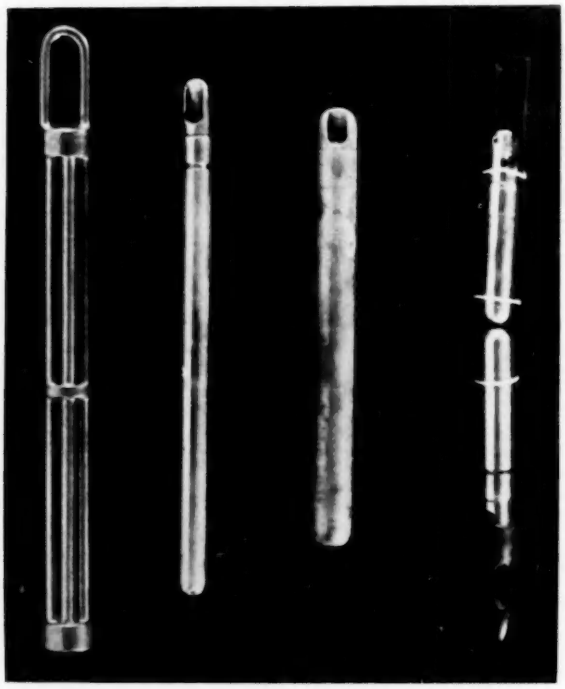


FIG. 4 — (Pressman and Emery) — Various containers for intertracheobronchial radium therapy.

a preliminary bronchoscopy. Features to be considered are the length of the tumor, its position in the tracheobronchial tree, the side involved, the contemplated dosage of radium, and, as I shall shortly dwell upon, the degree of preliminary pneumothorax which has been instituted.

The principle of tubes numbers 2 and 3 is simply that of a hollow, aluminum container with a screw cap. They are approximately 10 centimeters long and 7 millimeters in diameter. Radium needles in varying numbers are inserted into their lumen in tiers separated from each other by cotton. The needles themselves are of platinum of either one-half or one and one-half mm. thickness. This tube is primarily for the right main stem bronchus, and when inserted occupies a position such as is shown in Figure 5 filling the bronchus from its lowermost depths to well above the bifurcation.

FIG. 5 — (Pressman and Emery) — A solid aluminum container in situ in the tracheobronchial tree.

Under certain circumstances it is necessary that during the period of installation air be allowed to pass freely into the lung. This is absolutely impossible with the solid type container shown in Figure 5. Accordingly, there has been designed by Dr. Clyde Emery a tube which has the general appearance of an elongated birdcage. It consists of a series of hollow struts supported by thick circular bands of metal, one in the center and another removable band or collar at each end. The radium needles are placed in the lumen of the hollow struts and retained in position with the screw collar. The loop at the end is for the purpose of bronchoscopic removal of the tube. This is a very efficient apparatus, inasmuch as its bulk is sufficiently great so that it is almost impossible to displace it by the most severe paroxysm of coughing. It also permits close approximation of the radium to the bronchial

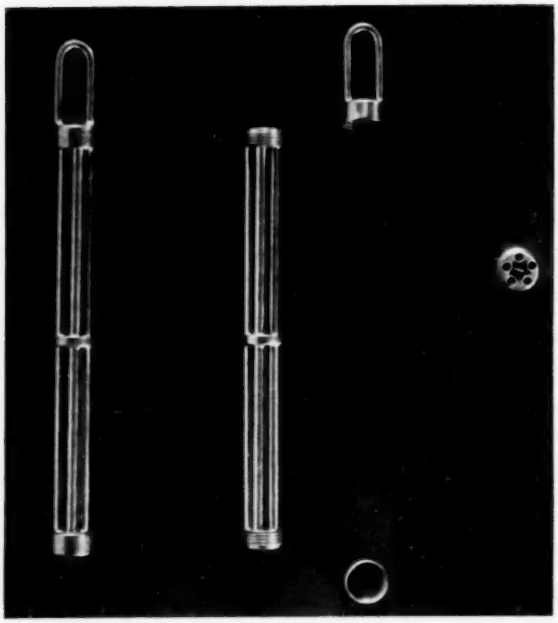


FIG. 6 — (Pressman and Emery) — The "Birdcage" container. In the central figure the collar on each end has been unscrewed. At the right is an end-on view with the collars removed. Radium needles are inserted into the struts through the five small holes. Both ends are alike.

wall and an even distribution of radium emanations throughout the length and circumference of the bronchus. When conditions warrant, the use of this tube is my method of choice.

Figure 7 demonstrates its appearance lying in situ in the tracheobronchial tree. The circle surrounding the site of early carcinoma is superimposed, so that we may see how long a segment of tracheobronchial tree above and below the lesion is irradiated. The importance of this cannot be stressed too often.

We come now to an entirely different type of applicator. This is shown lying in situ in the right main stem bronchus. The distal portion of a rubber catheter is used as an outer jacket. For purposes of demonstration half the diameter of the catheter has been removed, but, of course, this is not true when the catheter is in actual use. Within the catheter are placed two or even three very short aluminum containers. This produces the effect of a segmented tube with great pliability. It is especially useful in the left main stem bronchus where the relatively greater acute angle at

FIG. 7 — (Pressman and Emery) — The "Birdcage" container lying within the tracheobronchial tree. The site of most early carcinomata is demonstrated by the white circle.

which the bronchus comes off the trachea makes the use of a rigid tube difficult.

The question of radium dosage is best left to the radio therapeutist. This will vary in each case, depending upon the extent of the area over which the radium is to be distributed. The large birdcage, for example, has a capacity of 50 milligrams of radium and can be left in situ for 30 hours, making a total dosage of 1500 milligram hours. This is the largest dose we have ever employed. The relatively larger total dosage in this case was deemed safe because of the relatively large size of the area included within the field of radium activity. The smaller containers, such as those within the catheter, contain only 25 milligrams of radium and are left in situ for 40 hours for a total of 1000 milligram hours.

It is obvious that the insertion of so large a foreign body into the bronchus for so long a period of time must of necessity cause

FIG. 8 — (Pressman and Emery) — Short aluminum capsule-like containers lying in tandem within a catheter. Note that the butt ends face each other, otherwise the cap of the lower container would represent an area of non-radiation.

obstruction to the inflow of air with resultant massive collapse of the lung. This may be overcome in one of three ways:

(1) By maintaining the tube in situ for relatively shorter periods of time and repeating the insertion in order to make up the total dosage;

(2) By using hollow tubes of which the container nicknamed the "birdcage" is the best example, but even this tube after 15 to 20 hours may become occluded by thick mucopus or blood.

It does, however, represent a considerable improvement in this respect over the original solid container.

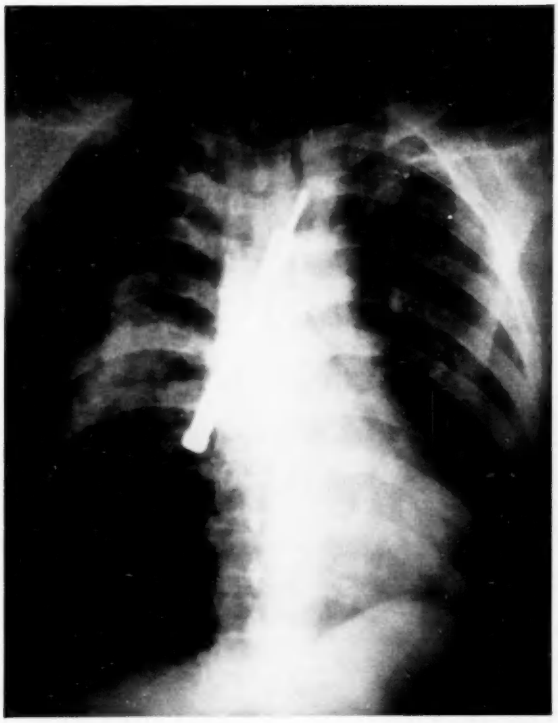


FIG. 9 — (Pressman and Emery) — The "Birdcage" container in situ in combination with pneumothorax.

The third method of preventing massive collapse is our present method of choice. At the suggestion of Dr. Jacob Abowitz, preliminary pneumothorax was instituted. This procedure wrote a new chapter in the development of the technique. The pneumothorax is made as complete as possible. We later found it advantageous to maintain this for many weeks following the radium insertion. It presents such outstanding advantages that its use has become routine except for those cases where adhesive bands prevent collapse from taking place. In those instances where it seems propitious, I deem it worth while to have the bands severed with the thoracoscope and pneumothorax then instituted.

The advantages of complete preliminary pneumothorax are many. The lung having been previously compressed, massive collapse cannot possibly occur either during the period of treatment or during the period of radium reaction, which comes six weeks

later. The collapse of the lung diminishes the possibility of hemorrhage. If the compression of the lung be sufficiently great, it is reasonable to assume that a greater area of lung tissue is brought into the field of effective radiation. The lung is put completely at rest so that movement of the container is less apt to occur. Obviously, if no air enters the lung, no effective cough can be produced from that side and the radium container is less apt to be coughed up. For these reasons I heartily endorse the use of preliminary pneumothorax as an integral and vital part of this method of treatment.

Figure 9 demonstrates the combined use of complete pneumothorax in conjunction with the insertion of a larger birdcage type of radium needle container. Under such circumstances the container can remain in situ almost indefinitely. Note the outline of the completely collapsed lung, the pneumothorax and the radium container.

The pre-operative preparation is of some importance. The patient is admitted to the hospital the day previous. That night two capsules of Nembutal or Amytal are administered for sleep. On the following morning prior to the operation, the patient receives 1000 to 1500 cc. glucose intravenously. An additional large dose of Nembutal is given and an hour later ¼ grain morphine and 1/150 grain of Scopolamine. These latter two are repeated just prior to insertion, if it seems necessary. I prefer to have the patient brought to the operating room in semi-coma.

The anesthetic is local as for any bronchoscopy except that one cc. of 10% cocaine is dropped into the involved bronchus.

Technique: First the bronchial lumen is reestablished by removing as much of the tumor as is necessary with either a punch forceps or fulguration. The technique of actual insertion varies according to the size of the capsule and the side involved. Some containers are difficult to remove and if this be anticipated, a long woven silk string is firmly tied to the proximal end, prior to the insertion. If the capsule is of sufficiently small diameter to fit through a bronchoscope, this is inserted and the container grasped with a foreign body forceps. It is then introduced into the bronchus through the bronchoscope and under direct vision carried as deeply into it as possible. This method is especially indicated in left-sided lesions. If the container is too large to fit through the bronchoscope, as is the case with the "birdcage," it is inserted through a direct laryngoscope. The larynx is exposed.

The container, seized in the grasp of a laryngeal alligator action forceps, is inserted between the cords. It is then carried down blindly until it has entered the bronchus, usually the right. The bronchoscope is then inserted and a bronchoscopic suction canula or foreign body forceps used to push the container deep into the bronchus, and its position checked by bronchoscopic vision. The string, if one has been used, is allowed to project from the mouth or nose and attached to the side of the face with adhesive tape. The string, however, tends to provoke cough, but since it is a safety factor, is probably well utilized. In some cases we use it; in others, we do not deem it necessary.

The patient is immediately taken to X-ray and the position of the tube checked fluoroscopically. If the patient can be made to cooperate, it is well to have him cough in order to determine whether or not a coughing paroxysm tends to displace the tube. He is then placed in bed in a sitting position with sideboards for protection. During the period of treatment, the patient is kept narcotized with frequent large doses of morphine. Fluids and caloric intake are provided by intravenous glucose. If coughing is profuse, which is not very often the case, provided morphine be given in adequate doses, or if it seems that copious amounts of mucus or pus are present, the tube is removed to be reinserted again after a day or two of rest. Divided doses are possible, but usually the tube can be maintained in situ for the entire calculated period. Occasionally the patient coughs up the smaller tubes, in which case it may block the larynx, so that vigilance on the part of the nurse is necessary. If this occurs she can easily remove the container by traction on the string, which is always used with tubes of small diameter. Intentional removal, if no string has been provided, is done through the bronchoscope with foreign body forceps. The post-operative course ordinarily is smooth and the patient remains quietly at home, or is slightly active for a period of about six weeks.

The pneumothorax is maintained by repeated installations of air as often as necessary. During this period, coughing and expectoration is apt to be increased. At the end of six weeks a radium reaction is to be anticipated. This occurs in the form of edema of the bronchus, which may be so extensive as to completely occlude it, or nearly so. The advantages of having maintained a pneumothorax are now apparent, since it prevents massive collapse of the lung, reduces the likelihood of lung sepsis and hemorrhage during the radium reaction. The temperature may

be slightly elevated, the cough becomes slightly more profuse and is productive of small or moderate amounts of yellowish gray sputum, sometimes foul. A certain amount of general debility occurs. This reaction is apt to be of several days' to a few weeks' duration, after which it gradually subsides. The pneumothorax is then allowed to absorb and the lung re-expands.

A word about complications: The obvious possible complications are hemorrhage, lung sepsis, pulmonary massive collapse and post-operative stricture of the bronchus. In about twelve cases we have had one massive collapse during treatment, but this occurred prior to the use of pneumothorax. One fatal hemorrhage developed eight weeks post-operatively. We have had no case of severe pulmonary sepsis, and no case of stricture of the bronchus sufficiently severe to require dilatation.

Concerning results following this method of treatment, I can say very little. It has been proven to my satisfaction that radium in adequate dosage, administered according to this technique, in properly selected early cases, can result in complete local disappearance of carcinomata of the bronchi. We feel for the following reasons that when radium is indicated, this is the method of choice, because (1) the technique is simple and relatively safe; (2) it provides adequate, carefully measured radium dosage; (3) radiation of equal intensity throughout all portions of the tumor, and (4) radiation of healthy tissue above, below and around it.

DISCUSSION

DR. LOUIS H. CLERF, Philadelphia: The radium applicators presented by Dr. Pressman are ingenious and overcome some of the objections to the types used earlier. They represent a definite improvement from the mechanical standpoint of radium application.

The results of irradiation therapy in bronchial carcinoma have not been gratifying whether X-ray was employed alone or in combination with interstitial radiation. For that reason the employment of radon seeds for interstitial radiation has been abandoned temporarily, at least, at the Radiological Clinic of the Philadelphia General Hospital. I am interested to know concerning the effects on the normal bronchial wall of these large doses of radium applied with little filtration. Is there extensive tissue destruction?

The outlook in cases of carcinoma of the main bronchus is absolutely hopeless so far as surgical treatment is concerned. Dr. Pressman is to be congratulated and should be encouraged in his endeavor to work out a technique of irradiation therapy that offers some hope to these patients.

DR. FRANCIS E. LEJEUNE, New Orleans: The increase in the incidence of carcinoma of the bronchus is attested by the frequent reports in the literature. While the increase in the number of cases reported may be partly due to more highly efficient diagnostic methods, the fact remains that pulmonary carcinoma forms about 1% of all cases at necropsy and more than 8% of all malignant diseases.

The treatment of carcinoma of the bronchus has been most unsatisfactory and all are agreed that surgery is the method of choice when possible. I must concur with Dr. Pressman that X-ray therapy has not been very encouraging in the treatment of bronchiogenic carcinoma. Kernan reports the treatment by X-ray therapy of 104 cases of carcinoma of the lung. While only 21 of this number received adequate therapy, the fact remains that only four patients survived a period of 15 to 28 months.

Heretofore radium has not been used advantageously in the treatment of bronchial carcinomata due to obvious difficulties. Dr. Pressman has overcome these difficulties and presented us with an excellent method for its use. Since bronchial carcinoma is inclined to metastasize fairly early, a permanent cure cannot be hoped for unless destruction of cancer tissue is accomplished early. An early diagnosis in carcinoma of the bronchus is imperative and must be followed by heroic treatment.

While I have not had an opportunity to try out the method advocated by Dr. Pressman, his reports convince me I shall use this method when the next opportunity presents itself. The contact of the radium with the growth and the irradiation of the surrounding tissue should be such as to warrant excellent results if the dosage is properly controlled.

Dr. Joel J. Pressman, closing: May I thank Dr. Clerf and you, Dr. LeJeune, for your very flattering but interesting discussions of this paper?

The only question that has been asked is on the effect on the normal bronchial wall of irradiation. The remote effects are chiefly a thickening of the fibrosis of the deeper structures. In the single case in which I have tissues, the epithelium appears darker than normally, with granules and apparently with loss of cilia. There is, however, a reestablishment of the columnar ciliated epithelium although it appears to be histologically not the same as the similar area of mucous membrane from the opposite main stem bronchus.

CLINICAL OBSERVATIONS ON BONE CONDUCTION*

W. J. McNALLY, M.D.
T. C. ERICKSON, M.D.; R. SCOTT-MONCRIEFF, M.D.;
D. L. REEVES, M.D.

MONTREAL

THE OBJECT of this research was to investigate bone conduction acuity in a series of patients suffering from inner ear or proven cochlear nerve lesions, such as acoustic nerve or other intracranial tumors. The patients examined were for the most part from the Montreal Neurological Institute and we are greatly indebted to Dr. Wilder Penfield and his associates for placing at our disposal this excellent material. Only those cases were included in which the intracranial lesion was proven at operation or at post-mortem examination.

A great difficulty in undertaking such a clinical investigation of hearing by bone conduction is the lack of uniformity in instruments, tests and methods for determining bone conduction. Much care was exercised in selecting the instruments. We were guided in our selection by the report of the Committee for the Consideration of Hearing Tests, of the Royal Society of Medicine, London, 1933, and by the report of the Committee on Tuning Forks and Tests of Hearing of the American Academy of Ophthalmology and Otolaryngology (1931). It was finally decided that the bone conduction of each patient should be tested by a variety of methods; tuning forks, the monochord of von Struychen, and by the audiometer bone conduction attachment, in order to be able to compare the results from each method.

* Research authorized by the Council and financed by the American Academy of Ophthalmology and Otolaryngology. From the Montreal Neurological Institute and the Department of Otolaryngology, Royal Victoria Hospital, Montreal. A résumé of these experiments was reported as part of a Symposium on Bone Conduction for the Detroit Meeting of the American Otological Society, 1936.

TUNING FORKS

In selecting tuning forks most suitable for this investigation, much valuable assistance was given us by Professor W. Watson of the Department of Physics, McGill University. The vibrations of each fork were compared and checked against the General Radio Oscillator No. 222. The vibrations from the prongs and from the stem of each fork were tested separately. The following forks were tested by this method and were all found to be quite true both for the prong and the stem vibrations: Edelmann's forks c^1, c^2, and c^3, an aluminum-magnesium alloy fork (Riverbank Laboratories) c^2, and Hartmann's c^1 and c^2 forks.

In spite of the fact that all of the above named forks proved to be accurate they were not all equally suitable for the carrying out of bone conduction tests. When struck a standard blow the Edelmann forks and the alloy fork had a considerable amount of overtones which were quite misleading and the vibrations from the prongs were so loud at the outset that they were easily heard by air conduction. Finally, the vibration period of these forks was so long that it became fatiguing for both patient and examiner by the time each test was repeated and checked a few times. The smaller Hartmann fork c^2 (512) which when tested proved to be accurate in its vibrations, was more suitable for our purposes in that it did not have as strong overtones, nor as strong an initial vibration, and finally the total length of vibration was not as long as in the larger forks and yet was long enough for accurate determination of the hearing both by air and bone conduction.

Two forks c^1 and c^2, made according to the specifications of the Hearing Test Committee of the Royal Society of Medicine, were ordered from London and arrived before these experiments were completed, but they were not found to be any more satisfactory for testing Bone Conduction than the Hartmann c^2 fork we were already using.

Considerable thought was given to the range of forks which should be used to test the bone conduction. The majority of otological writers now agree that forks below c (128 d.v.) and above c^3 (1024 d.v.) are totally unsuitable for bone conduction tests but there is considerable variation of opinion about the forks within this small bracket. See Clarke (1929), Hallpike (1930), and the Report of the Committee for the Consideration of Hearing Tests of the Royal Society of Medicine, London (1933). The majority of the writers favor either c^1 (256 d.v.) or c^2 (512 d.v.).

With a c^1 fork, it is impossible to be sure, that at threshold strength, the patient is not actually feeling rather than hearing the vibrations. On the other hand, for a c^3 fork the vibrations by air are so much more pronounced than the vibrations from the stem that it is possible that the sound may be heard more by air than by bone conduction. It was therefore decided that the fork c^2 (512 d.v.) was the most practical to use for routine bone conduction tests. This is supported by the Committee on Methods of Testing Hearing by Bone Conduction of the American Otological Society, 1936.

The Weber test was recorded in each patient but instead of reporting the bone conduction tests in terms of Rinné and Schwabach it was thought more satisfactory to examine separately and record the air conduction, the bone conduction and the absolute bone conduction times for each ear in each case. In order to do this a standard method of activating the fork must be employed and the tests should be done under similar conditions each time.

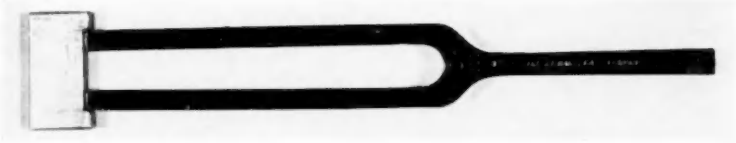


FIG. 1 — (McNally) — Hartmann tuning fork c^2 (512) with brass clip attached for activating the fork.

A reasonably efficient method of activating the fork was adapted from the Physiology Laboratory, McGill University. A small brass clip (see Fig. 1) was made which just slips over the ends of a given tuning fork when the end of the prongs are slightly compressed. When the clip is suddenly pulled off, the fork vibrates with a minimum of overtones and always for the same length of time, with a variation of only a few seconds. For the Hartmann fork c^2 (512), thus activated, the duration of the vibration as heard by air conduction in a quiet room is about 70 seconds. That is an average for several normal examiners.

Care was taken to establish a routine method of handling the tuning forks in order to standardize such factors as the rate of decay from the damping effect of the cranial bones, etc., as pointed out by Clark (1929) and re-affirmed by the Committee for the Consideration of Hearing Tests of the Royal Society of Medicine, London, 1933.

THE MONOCHORD

The von Struychen monochord used in these examinations was tested and accurately set by checking it with the General Radio Oscillator No. 222. Cotton wool saturated in methyl alcohol was used to stroke the wire. The longitudinal or double vibrations were the only ones tested. The monochord was used to test the upper tone limit of both air and bone conduction.

THE AUDIOMETER BONE CONDUCTION ATTACHMENT

We used the Bone Conduction attachment of the 2A Western Electric Audiometer. The instrument was checked by the Northern Electric Company during the course of these experiments and was carefully examined before each test to assure its proper working. It was decided that on the audiometer in addition to testing for the note c^2 we should also test for the note c^1 in every case in the hope of showing some variation between the two. Knudsen and Jones (1931) used the Bone Conduction receiver of the audioamplifier and tested Bone Conduction over a range from 64 d.v. to 4096 d.v. Both ordinary or relative bone conduction and absolute bone conduction were tested by the audiometer.

We were fully aware of the influence of the surroundings upon the accuracy of any hearing test. Because of the serious condition of many of these patients, they could not be removed from the wards so that a majority of the tests were carried out in a relatively noisy ward. When possible or where it seemed to be especially indicated the test was repeated in a quiet room. The room used for these retests was particularly quiet but was not sound-proofed. Insofar as possible the results in each case have been interpreted in the light of the surrounding conditions as well as with reference to the age (see Fig. 2) and the physical and mental condition of the patient. Slight individual differences between the examiners and the variability of the test methods themselves were also considered. Every care was taken in carrying out the different tests to reduce the variables to a minimum. In almost every instance each examination was carried out in the presence of at least two and usually three of the examiners and all abnormal findings were checked and rechecked by each examiner present. An impression of the case was recorded at the end of each examination.

It was felt from the outset that an examination of the bone conduction could not be properly interpreted unless it was accom-

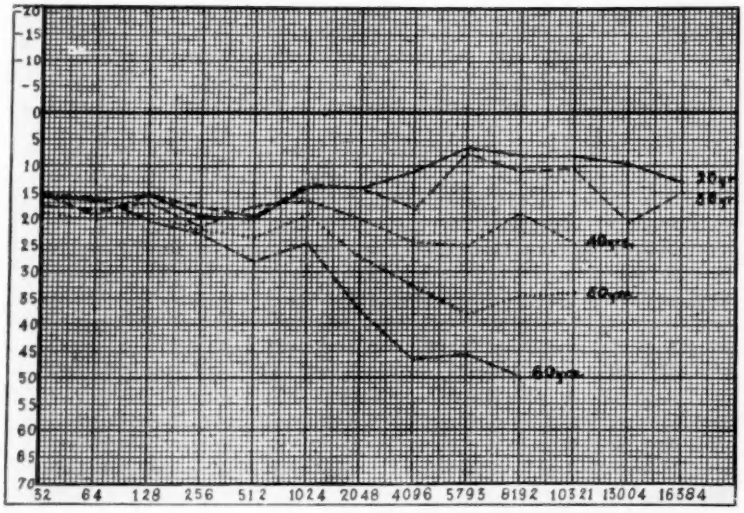


FIG. 2 — (McNally) — Audiogram showing the means for the different age groups (Bunch 1929).

panied by a very careful audiogram and by air conduction tests by the tuning forks and the monochord. The vestibular tests were also done as an added check and help in interpretation.

Because it is so difficult to standardize the voice and watch tests they were not included in the routine examination. At the end of the investigation, however, it was felt that had we included them this would have been an excellent opportunity of comparing their value in diagnosis with the more mechanical tests.

METHOD OF EXAMINATION

A suitable routine of examination was arrived at and was used in all cases except where the preliminary hearing tests had been done before this investigation was undertaken. Insofar as possible a complete hearing test and vestibular examination was done before and after any operative procedure.

The complete examination consisted of:

(a) A careful history and inspection of the ears, nose and throat.

(b) An audiogram (2A Western Electric Audiometer).

(c) Tests with the Audiometer Bone Conduction Apparatus of bone conduction and absolute bone conduction for the notes 256 d.v. and 512 d.v.

(d) Tests with a tuning fork (512 d.v.) of air conduction, bone conduction and absolute bone conduction.
(e) Tests with a von Struychen monochord of air conduction and bone conduction.
(f) A vestibular examination.
(g) Masking of the non-tested ear for any of the above tests when indicated.
(h) Impression of the case.

An effort was made in every case to determine the exact condition of the middle and internal ears in order that loss of hearing would not be attributed to an intracranial lesion when it could be explained by local ear disease.

THE ABSOLUTE BONE CONDUCTION TEST

A valuable part of the bone conduction examination is the absolute bone conduction (ABC) test. In the present investigation it was done in connection with the fork tests and with the audiometer bone conduction tests. It consists of closing the external canal while the stem of the fork is held against the mastoid. When carried out on a normal person in an ordinary room, absolute bone conduction is twice as long as ordinary (relative) bone conduction and is about half as long as air conduction. These ratios vary with the surrounding noise. The routine use of this test was advocated in a report of the Committee of Research of the Royal Society of Medicine, London, in 1917. However, it never received general adoption and Hastings (1929) suggests that the reason may be that this report appeared during the war and may have been overlooked. Clarke (1929) states that the test was described as early as 1883 by Pomeroy. The test and its advantages are described in detail by Hastings (1929), Clarke (1929), Hallpike (1930), and by the Committee for the Consideration of Hearing Tests of the Royal Society of Medicine, London, (1933). Very similar to the absolute bone conduction test is a modification of the Weber test described by David-Galatz (1934).

Hallpike's explanation of the phenomenon is that closing the external canal shields the internal ear from room noises and allows the tuning fork against the mastoid to be heard for a longer time, or for the same length of time that it would be heard in a quiet or sound-proof room. A patient who has a middle ear lesion and a normal internal ear on the same side has a longer relative

bone conduction than normal because the damaged middle ear shields the internal ear from the room noises just as closing the external canal would do. Hallpike (1930) found that patients with middle ear lesions and increased relative bone conduction, when examined in a sound-proof room, or when compared with the examiner's absolute bone conduction, have bone conduction comparable with the examiner's provided there is no internal ear lesion. See also Knudsen and Jones (1931), and Kobrak, Lindsay and Perlman (1935).

This test is particularly valuable when an examination must be carried out in the ward because it offsets the noise factor in both the patient and the examiner.

The ABC test carried out in the following manner may give additional information as to which internal ear is really hearing a bone conducted sound in the case of an asymmetrical hearing lesion. The fork is placed on one mastoid and both ear canals are closed simultaneously and the ABC time is recorded. With the fork on the same mastoid, the ABC time is recorded while only the ear canal on the same side is closed, and finally the test is repeated while the ear canal on the opposite side is closed. This may be illustrated by presuming a case with right sided nerve deafness. Ordinary or relative bone conduction may be normal or even lengthened. When both canals are blocked, bone conduction over the right mastoid may be equal to the examiner's absolute bone conduction. On the other hand, when only the right canal is blocked the hearing for the fork is not increased above the time of relative bone conduction for this ear. At this point, the left ear canal is blocked and the time for hearing the fork on the right mastoid increases and may equal the absolute bone conduction of the better hearing left ear. In the absence of masking this simple test is an indication that the apparent bone conduction over the right mastoid is really bone conduction being heard in the more normal left ear. The bone conduction is improved when the left ear is screened from room noises, but on the other hand bone conduction is not increased by closing the right or deaf ear. The value of the test can be checked by masking both ears alternately. It is found that in such a case applying the masker to the right ear has no effect on the bone conduction time over the right mastoid, whereas when the masker is applied to the left ear, bone conduction over the right mastoid is no longer heard. Examples of the value of this method of carrying out the absolute bone conduction are seen in cases 43 and 48.

MASKING

In selecting a suitable masking apparatus we were guided by an excellent description of masking by Bunch (1931). Occasionally a Barany noise apparatus was used but for the most part we used a continuous current of air or else an electrical buzzer of the door-bell type, a modification of that described by Bunch. A resistance was inserted to give variation of the loudness. Instead of having a telephone receiver the small buzzer itself was enclosed in a rubber sponge and was held in contact with the patient's ear.

As a source of a continuous current of air the stream from an ordinary compressed air circuit was used, and directed into the ear canal. The strength and noisiness of the current could be varied from the outlet valve. It was felt that when used with sufficient force to mask some sounds the air was so cold or so disagreeable as to be acting as an irritant rather than as a simple masker of the sound in the non-tested ear (see case 5).

Since we were testing bone conduction with the audiometer bone conduction attachment, with the forks and with the monochord, masking was not quite the same problem in each case. The electric buzzer appeared to be very ineffective as a masker for the monochord. In fact, it was only with the strongest current of air that the monochord could be masked out in a few cases where there was proven nerve deafness. In one case, for instance, where the right eighth nerve had been cut (case 48) the monochord BC on the right could not be masked out with the electric buzzer and only with the very strongest current of air could it be eliminated. The monochord and especially monochord bone conduction in cases of unilateral nerve deafness may be most misleading and very difficult to check up even with masking. Possibly if a masker of somewhat similar high frequencies to the monochord itself were used masking might be more effective.

Bunch (1931) refers to the fact that in some normal individuals masking occasionally increases hearing for some notes in the non-masked ear. The phenomena is probably similar to the Crossed Paracusis of Hastings and Scarff (1928). In this investigation it was noted in a few cases (cases 41 and 42) that attempting to mask the monochord actually increased the hearing by BC in the non-masked ear.

These results, however, support those of other workers in stressing the importance of masking in cases of asymmetrical or unilateral deafness. As pointed out by Bunch, discrepancy in

the two ears, especially if there is a shadow curve audiogram, calls for masking as shown by cases 42, 43, 44, 48, and Figs. 11, 12, 16 and 17. Furthermore, where there is a high grade bilateral deafness, masking may bring out some unsuspected differences between the two ears (see case 32, Figs. 7 and 8).

In doing the absolute bone conduction test, if blocking the opposite ear canal is more effective in prolonging the hearing by bone conduction, masking the opposite ear as previously mentioned will also support the idea of nerve deafness in the tested ear.

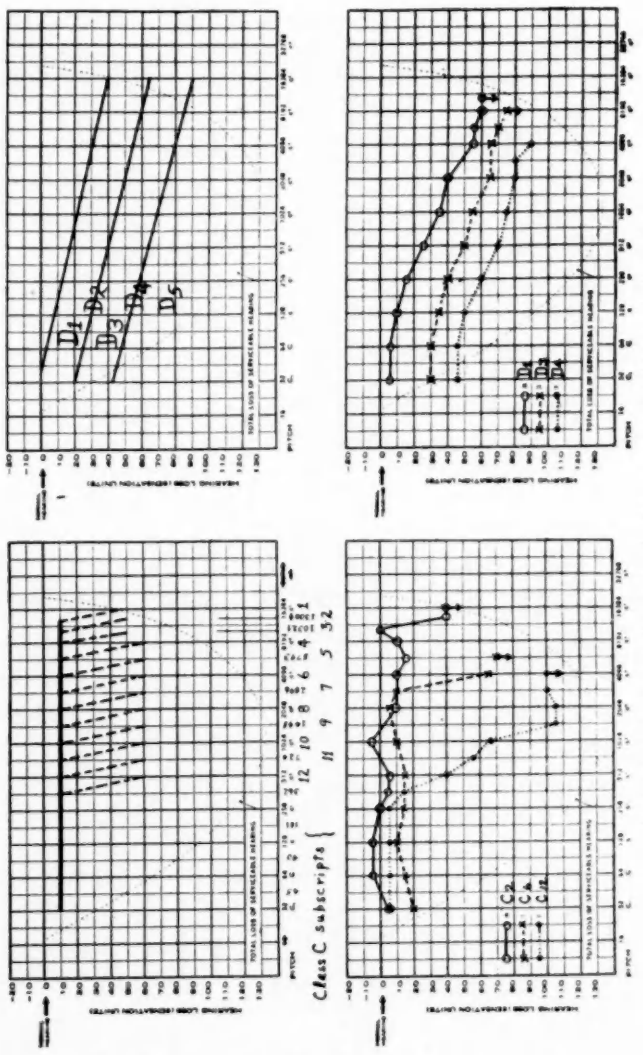
A word of explanation is in order with reference to certain abbreviations which have been used, especially in the preparation of the tables in the case reports. Masking with a current of air is designated by A.M., masking by the Barany noise apparatus by B.M., and masking by the electric buzzer as E.M.; s.u. stands for sound units, and d.v. for double vibrations.

CLINICAL MATERIAL

During the present investigation fifty patients from the Neurological Institute were studied. The cases were chosen either because the intracranial condition had been determined by a previous operation, or because they were about to be operated upon, or else because they were to be subjected to some special procedure.

In order to make this survey of most use to those specially interested in the subject, it was decided to report the complete hearing examination in every instance together with a brief description of the intracranial diagnosis and operative procedures. We present our conclusions drawn from this material but it is possible for anyone to review the material and make their own deductions.

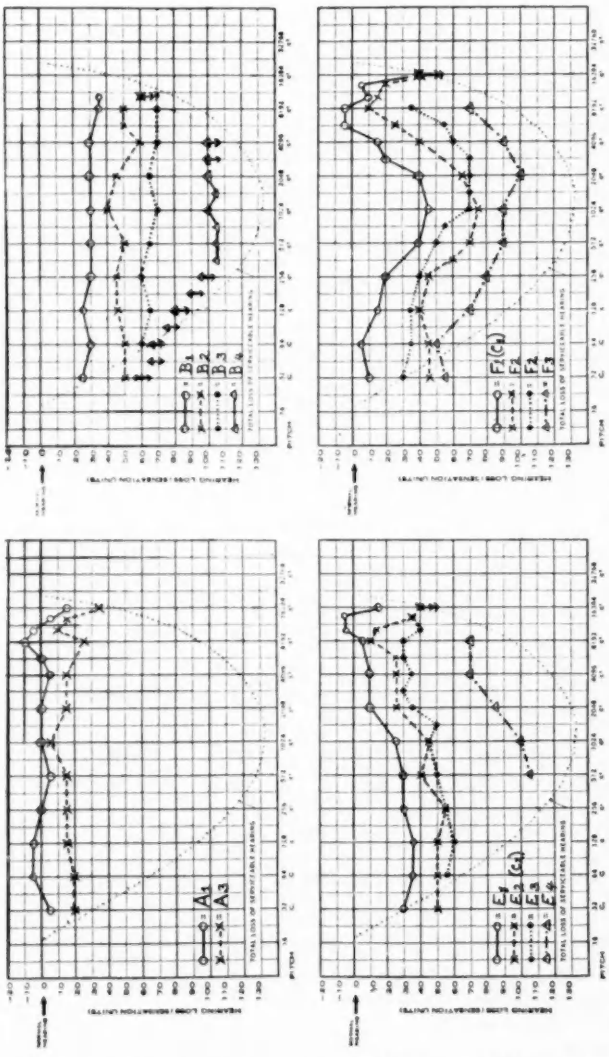
Reproducing all the audiograms seemed unnecessary, but those not reproduced are described according to the excellent classification published by Guild (1932). In order to help the reader visualize each audiogram, his diagrams are reproduced for ready reference (Figs. 3 and 4). The audiograms in this series requiring special description or those of special cases have been reproduced. In these audiograms, circles joined with a continuous black line always indicate the right ear, while the left ear is always indicated by the letter X and an interrupted black line. When masking is used a filled-in circle represents the right ear when the left ear is masked, and a filled-in X represents the left ear when the right ear is masked. In order to avoid any misunder-



Actual examples of audiograms which have been classified as indicated.

FIG. 3 — (McNally) — Examples of audiograms classified by Guild (Guild 1932).

standing the significance of each of these characters is given on the individual audiogram and the type of masking is specified in each case. When a note is not heard at the full strength of the audiometer, the character representing the deaf ear is shown on



Left: Diagram showing the twelve possible subclasses of Class C audiograms, and actual examples of three which have been classified as indicated.
Right: Copy of the ruled chart used in determining the subclassifications to be given audiograms of Class D, and actual examples of three which have been classified as indicated.

FIG. 4 — (McNally) — Further examples of audiograms explaining Guild's method of classification (Guild 1932).

the audiogram at the point of maximum loudness of the machine with a down-pointing arrow attached to the character (see Fig. 6).

The cases have been divided into six groups for purpose of description and each group will be dealt with separately.

GROUP ONE

Alteration of Intracranial Pressure

When carrying out an otolaryngological examination in a patient suffering from possible intracranial disease, one is called upon frequently to estimate the possible influence of changes of intracranial pressure upon the hearing and vestibular mechanism. To obtain accurate information about the effect of intracranial pressure changes, a hearing test must be done immediately before and after a known pressure change. The present investigation afforded an opportunity to make some observations upon this question, because many of the patients in a neuro-surgical service have either an encephalogram or a ventriculogram done as a diagnostic procedure.

An encephalogram consists of the withdrawal of approximately 90 cc. of spinal fluid by lumbar puncture, while the patient is sitting up, and its replacement by approximately 110 cc. of oxygen. What happens to the intracranial pressure is not exactly known but the air in the intracranial spaces remains for two or three days or more and the subjective disturbances persist for the same length of time. Usually when a second encephalography is attempted in 24 hours' time the pressure is found to be low.

Ventriculography consists in the direct replacement by ventricular puncture of 20 or 30 cc. of ventricular fluid with a similar quantity of oxygen.

From these procedures it is presumed that there is a very definite alteration of intracranial pressure relationships. Aird (1936) injected various gases in dogs and recorded the spinal fluid pressure changes. The injection was as done for encephalography. There was great variation, low as well as high pressures being recorded. He found that these changes occurred up to 24 to 48 hours after the injection of the various gases. The majority of cases suggested a rise of spinal fluid pressure.

From the above observations it would seem reasonable to presume that there are intracranial pressure changes within the first 24 hours following encephalography and ventriculography.

With these ideas in mind, ten patients were examined immediately before encephalography and within 24 hours after and three patients were examined just before ventriculography and the test repeated within 24 hours afterwards.

CASE REPORTS

Case No. 1 — Miss M. B., age 30. Admitted March 17, 1936.

Diagnosis: Jacksonian Epilepsy, focal area of cortical atrophy.

Note: The epileptic seizures were characterized by micropsia, dizziness, tinnitus, pain in the left ear, and numbness in the right hand.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 18, 1936

NOSE and THROAT — Normal. Post. cerv. glands of both sides nodular.

OTOSCOPY — Drums normal. Left ear canal narrower than the right. The patient has had some pain in the region of the left ear, which becomes a definite noise before an attack.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	50 s.u.	55 s.u.
c^1 (256)	BC	75 s.u.	75 s.u.
	ABC	50 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	15 seconds	18 seconds
	ABC	28 seconds	30 seconds

Weber — BC not referred while ABC tends to be referred to the left side.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION

Cold Caloric Test — Normal. (The patient stated that the dizziness which she experienced was exactly like that preceding her attacks. She became very much frightened, thinking she was about to have an attack.)

Operative Procedure: March 20, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 21, 1936

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	75 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	75 s.u.	65 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	60 seconds
	BC	22 seconds	22 seconds
	ABC	40 seconds	50 seconds

Weber — Slight tendency to be referred to the right when the fork is placed in the midline with both ears blocked, otherwise is not unusual in either BC or ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal reactions.

OPERATIVE PROCEDURES:

April 9, 1936 — Operator, W. G. Penfield. Left posterior osteoplastic craniotomy and excision of cerebral cicatrix. Near the midline in the mid-parietal region, the convolutions were removed over an area of 3x3 cm. and down to a depth of 4 cm. to the falx.

May 6, 1936 — 30 days after operation, the patient has recovered quite satisfactorily from the operative procedure. General condition excellent, and she is free from attacks.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 6, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal as before operation.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	55 s.u.	55 s.u.
c^3 (256)	BC	70 s.u.	70 s.u.
	ABC	45 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	25 seconds	25 seconds
	ABC	45 seconds	42 seconds
Weber — ABC and BC equal.			
Monochord	AC	18,000 d.v.	18,000 d.v.
	BC	20,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal reactions.

IMPRESSION: The Jacksonian attacks included dizziness and tinnitus, and yet removal of the cortical area which on stimulation reproduced these symptoms had no demonstrable effect upon the hearing and vestibular tests one month after the operation, although there may have been some effect immediately after the operation.

Cortical stimulation of the mid-line, mid-parietal area elicited dizziness from this patient (Foerster's area, Fig. 5).

Encephalography had no effect upon the hearing or vestibular tests twenty-four hours after the procedure.

CASE No. 2 — Mrs. Y. B., age 30. Admitted March 24, 1936.

DIAGNOSIS: Psychoneurosis, hysterical type.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 28, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	65 s.u.	60 s.u.
	ABC	45 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	70 seconds
	BC	20 seconds	22 seconds
	ABC	35 seconds	32 seconds

Weber — Not referred for BC or for ABC.

Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: April 1, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 2, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Drums are unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	75 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	40 s.u.	40 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	25 seconds	25 seconds
	ABC	35 seconds	35 seconds

Weber — Not referred for BC or for ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has not altered the hearing in either ear.

CASE NO. 3 — Mr. T. H., age 32. Admitted March 3, 1936.

DIAGNOSIS: Epilepsy, grand and petit mal, etiology undetermined.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 4, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Drums normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear, but with dip at 4096 d.v. in left ear down to 45 s.u. (Similar to Fig. 9).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	55 s.u.	50 s.u.
c^1 (256)	BC	65 s.u.	65 s.u.
	ABC	40 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	60 seconds	60 seconds
	BC	20 seconds	20 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	18,000 d.v.	18,000 d.v.
	BC	19,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 7, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 8, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Essentially the same as before operation.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	65 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	70 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	62 seconds
	BC	23 seconds	20 seconds
	ABC	40 seconds	43 seconds

Weber — Not referred for BC or for ABC.

Monochord	AC	18,000 d.v.	18,000 d.v.
	BC	20,000 d.v.	21,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography caused no change in hearing.

CASE No. 4 — Mrs. N. L., age 37. Admitted March 10, 1936.

DIAGNOSIS: Epilepsy, grand and petit mal, etiology not determined.

NOTE: Repeated encephalograms and one ventriculogram revealed nothing of localizing value. Duration of the symptoms three years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 11, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 right; A_2 left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	70 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	60 s.u.	65 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests

c^2 (512)	AC	65 seconds	65 seconds
	BC	20 seconds	20 seconds
	ABC	25 seconds	25 seconds

(Under these conditions, BC = 20 seconds, and ABC = 45 seconds normally)

Weber — The fork tends to be referred to the left when it is placed almost anywhere on the vertex. On the other hand, if it is placed on the teeth or glabella, it tends to be referred to the right. When both ears are blocked, the fork tends to be referred to the left from the vertex, but if moved towards the right ear, it tends to be referred to the right.

		RIGHT	*LEFT*
Monochord	AC	17,000 d.v.	16,000 d.v.
	BC	18,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 12, 1936. Encephalography.

NOTE: This patient was examined 24 hours after the first encephalography, but not after any of the others.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 13, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 right; A_2 left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	80 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	65 s.u.	65 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests

c^2 (512)	AC	70 seconds	70 seconds
	BC	23 seconds	23 seconds
	ABC	35 seconds	35 seconds

Weber — The fork was referred to the right ear from any point on the skull, even when placed over the left mastoid. When both ears are blocked, the fork is referred to the right ear from the midline, but if moved to the left of the midline, it is heard in the left ear.

		RIGHT	*LEFT*
Monochord	AC	16,000 d.v.	17,000 d.v.
	BC	17,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: A definite diagnosis of an intracranial lesion could not be established in this patient. The hearing tests carried out before and after encephalography were roughly within normal limits. We have no suggestion to offer in explanation of the slight diminution of absolute bone conduction for the fork c^2 (512). The peculiar reference of the Weber apparently has no explanation when considered in the light of all the other findings. It may serve in conjunction with many other examples to show how misleading this test may be.

CASE No. 5 — Mr. C. S., age 54. Admitted March 1936.

DIAGNOSIS: Cerebral thrombosis, middle cerebral artery, left, old standing. Bilateral chronic suppurative otitis media, right active, left healed. General arteriosclerosis.

NOTE: Patient had discharging ears in childhood.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 12, 1936

NOSE and THROAT — Large injected cryptic tonsils.

OTOSCOPY

Right Ear — Canal filled with foul-smelling discharge. Whole of the drum is destroyed except for Schrapnel's membrane. The mucosa of the middle ear is much injected.

Left Ear — Large central perforation occupying the whole of the posterior half of the drum. It has been healed over by a very thin membrane which is much retracted. The anterior part of the drum is intact though covered with a large calcium plaque. Healed chronic suppurative otitis media.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right D_4; Left F_1 C_6 down to 50 s.u.

(On masking left ear with stream of air, right ear drops to D_3)

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with A.M.	No masking	Op. ear masked with A.M.
c^2 (512)	BC	60 s.u.	75 s.u.	65 s.u.	N.H.
	ABC	55 s.u.		55 s.u.	
c^1 (256)	BC	45 s.u.	60 s.u.	50 s.u.	75 s.u.
	ABC	40 s.u.		50 s.u.	
Tuning Fork Tests					
c^2 (512)	AC	7 seconds		45 seconds	
	BC	23 seconds	23 seconds	25 seconds	
	ABC	27 seconds		30 seconds	

Weber — Not referred for either BC or ABC.

Monochord				
	AC	0		12,000 d.v.
	BC	14,000 d.v.		14,000 d.v.

NOTE: For bone conduction, masking the right or poorer hearing ear caused a marked lessening of hearing for bone conduction in the left or better ear, whereas masking the left or better hearing ear had much less effect upon the hearing for bone conduction in the right or poorer hearing ear. The only explanation we could give for this was the stream of cold air was more disturbing to the actively suppurating right ear. The discomfort so distracted his attention that he no longer heard with the left or better hearing ear.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 14, 1936 — Encephalography.

POST OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 16, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right D_4; Left B_1 C_6 to 75 s.u.

(The effect of masking the left ear with stream of air for note 2048. Hearing, right ear, for this note dropped 35 s.u.)

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with A.M.	No masking	Op. ear masked with A.M.
c^2 (512)	BC	50 s.u.		50 s.u.	
	ABC	45 s.u.	70 s.u.	45 s.u.	
c^1 (256)	BC	45 s.u.	45 s.u.	45 s.u.	70 s.u.
	ABC	40 s.u.		40 s.u.	
Tuning Fork Tests					
c^2 (512)	AC	10 seconds		45 seconds	
	BC	30 seconds	22 seconds	37 seconds	8 seconds
	ABC	50 seconds		25 seconds	
		43 seconds		32 seconds	
		35 seconds		30 seconds	
Weber — Not referred for BC or ABC.					
Monochord	AC	10,000 d.v.	N.C.	12,000 d.v.	N.C.
	BC	14,000 d.v.	N.C.	15,000 d.v.	N.C.

NOTE: For the bone conduction tests, as noted before, masking the right ear caused a greater loss of hearing in the left than the reverse. At this examination, the fork tests are more in keeping with a double middle ear lesion. BC is relatively increased and more nearly approximates ABC.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: There has been very little change in the hearing tests before and after encephalography. There is an obvious bilateral middle ear lesion. It is interesting to note that the audiogram would suggest a high grade nerve deafness in the right ear, whereas the bone conduction tests and the monochord tests suggest very little nerve deafness in either ear.

A most difficult finding to understand is that masking with a stream of cold air, when applied to the supposedly poorer hearing ear, caused a greater loss of hearing for bone conduction in the supposedly better hearing ear than the reverse. Presuming that this might be due to some type of pain phenomenon from the cold air douche acting upon the actively suppurating right ear, it was decided to repeat the tests using a graduated electrical buzzer as the masking apparatus.

CONTROL EXPERIMENT

USING THE GRADUATED ELECTRICAL BUZZER — May 11, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right D_4; Left B_1 C_6 down to 65 s.u.

(Masking either ear with electric buzzer at full strength caused very little reduction in the opposite ear.)

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with E.M.	No masking	Op. ear masked with E.M.
c^2 (512)	BC	55 s.u.	65 s.u.	60 s.u.	65 s.u.
	ABC	55 s.u.		55 s.u.	
c^1 (256)	BC	45 s.u.	60 s.u.	45 s.u.	60 s.u.
	ABC	40 s.u.		45 s.u.	

NOTE: Masking the homolateral ear gave roughly the same results as those in which the contralateral ear was masked.)

Tuning Fork Tests					
c^2 (512)	AC	10 seconds		40 seconds	
	BC	32 seconds	25 seconds	37 seconds	23 seconds
	ABC	42 seconds		40 seconds	

Weber — Not lateralized for BC or ABC.

Monochord					
	AC	11,000 d.v.	10,000 d.v.	12,000 d.v.	11,000 d.v.
	BC	15,000 d.v.	15,000 d.v.	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not repeated.

FINAL CONCLUSION: Masking the left ear on the audiometer with an electrical buzzer confirms the presence of a nerve deafness in the right ear, but the drop in the right ear is less than that caused by the use of a stream of air. Use of the electrical masker in connection with the bone conduction tests would suggest a bilateral middle ear deafness, bone conduction being well preserved in both ears and ABC approaching BC.

In spite of the great loss of air conduction in the right ear as compared with the left ear (bone conduction being equal in both) the Weber test has not been referred to the right ear as one would expect.

Whereas, masking either ear with a current of air eliminated hearing for the monochord in the opposite ear, masking with an electric buzzer had no effect upon the monochord.

CASE No. 6 — Mr. C. S., age 23. Admitted March 16, 1936.

DIAGNOSIS: Peripheral facial paralysis, right side.

NOTE: Sudden onset 14 months previously (cause undetermined).

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 17, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_1 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	45 s.u.	45 s.u.

Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	35 seconds	35 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for BC or for ABC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	19,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 19, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 21, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_1 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	65 s.u.	65 s.u.
c^1 (256)	BC	50 s.u.	50 s.u.
	ABC	40 s.u.	40 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	35 seconds	35 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for AC or for BC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	19,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has caused no alteration of hearing function.

CASE No. 7 — Mr. R. S., age 20. Admitted March 16, 1936.

DIAGNOSIS: Epilepsy, grand mal, idiopathic.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 17, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_1 for each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	45 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	40 s.u.	40 s.u.
Tuning Fork Tests			
c^2 (512)	AC	80 seconds	80 seconds
	BC	35 seconds	35 seconds
	ABC	50 seconds	50 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 28, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION
March 30, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_1 for each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	40 s.u.	40 s.u.

Tuning Fork Tests

c^2 (512)	AC	75 seconds	75 seconds
	BC	32 seconds	30 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has not caused any appreciable alteration in the hearing.

CASE NO. 8 — Mr. A. S., age 47. Admitted March 7, 1936.

DIAGNOSIS: Anxiety neurosis. Chronic suppurative otitis media, right, healed.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION
March 10, 1936

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Shows a thinly healed central perforation involving the whole of the posterior-inferior quadrant. The postero- and antero-superior quadrants are each covered with calcium plaques. The membrane of Shrapnel's area is retracted, but intact. There is no evidence of active disease in this ear. X-rays show the mastoids to be small and sclerosed, and the sinuses appear clear.

Left Drum — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.

c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	50 s.u.	55 s.u.

Tuning Fork Tests

c^2 (512)	AC	65 seconds	65 seconds
	BC	23 seconds	25 seconds
	ABC	35 seconds	37 seconds

Weber — From practically all points it is referred to the right ear, including teeth and glabella. ABC from the midline is not referred. From either side, including the left mastoid, it is referred to the right. Blocking the left ear refers all sounds to the left, including the right mastoid.

		RIGHT	*LEFT*
Monochord	AC	15,000 d.v.	14,000 d.v.
	BC	15,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 13, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 14, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	80 s.u.
	ABC	65 s.u.	65 s.u.
c^1 (256)	BC	60 s.u.	70 s.u.
	ABC	50 s.u.	55 s.u.

Tuning Fork Tests

c^2 (512)	AC	60 seconds	60 seconds
	BC	20 seconds	18 seconds
	ABC	30 seconds	35 seconds

Weber — Is not consistently referred to either ear. When both ears are closed; the fork in the midline is referred to the left ear, but if moved to the left about 2 inches it is referred to the right ear. If the fork is placed over the left mastoid and the ears not blocked, it is referred to the right ear; if the ears are blocked it is referred to the left ear.

		RIGHT	*LEFT*
Monochord	AC	13,000 d.v.	14,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

Watch Test — 20/30 in each ear.

Whispered Voice — Repeated at 20 feet in each ear.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has had no pronounced effect upon the hearing tests. It is worthy of note that even though the audiometric and fork tests of bone conduction show very little if any change from normal, and show very little difference on the two sides, the Weber does tend to be referred to the right, which is the side of the old middle ear disease. On the other hand, the Weber test itself is not consistent in that from the vault, the fork was not referred to the right ear, whereas from the left mastoid, it was referred to the right. As has been previously stated none of these findings was constantly present. This is another instance pointing to the great care necessary in attempting to assess the clinical value of the Weber test. Any slight hearing loss in this patient may be explained by the old middle ear disease.

CASE No. 9 — Mr. T. S., age 50. Admitted March 5, 1936.

DIAGNOSIS: Psychoneurosis, anxiety type.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 9, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear, but with a dip at 4096 d.v., left ear, down to 40 s.u. (Similar to Fig. 9).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	45 s.u.	50 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	40 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	60 seconds
	BC	25 seconds	20 seconds
	ABC	50 seconds	50 seconds

Weber — Not lateralized for BC or ABC.

Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: March 13, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 14, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_3 each ear, but with a dip at 4096 d.v., left ear, down to 40 s.u. (Similar to Fig. 9).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	45 s.u.	45 s.u.
c^1 (256)	BC	65 s.u.	65 s.u.
	ABC	45 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	25 seconds	25 seconds
	ABC	50 seconds	50 seconds

Weber — Not lateralized for BC or ABC.

Monochord	AC	14,000 d.v.	14,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has not caused any alteration of hearing.

Case No. 10 — Mr. H. T., age 48. Admitted March 14, 1936.

Diagnosis: Post-traumatic headache.

Note: History of head injury one and a half years ago followed by unconsciousness for four or five days.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 18, 1936

NOSE and THROAT — Small amount of mucopurulent material expressed from the tonsils on each side. Cervical glands are palpable, more on the right side than on the left.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	60 s.u.	60 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests			
c^2 (512)	AC	55 seconds	65 seconds
	BC	12 seconds	15 seconds
	ABC	30 seconds	32 seconds

Weber — Not definitely referred to either side for BC or ABC.

Monochord			
	AC	14,000 d.v.	14,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

Operative Procedure: March 24, 1936. Spinal subarachnoid insufflation of air.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 25, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	65 s.u.	60 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests			
c^2 (512)	AC	55 seconds	55 seconds
	BC	17 seconds	17 seconds
	ABC	28 seconds	28 seconds

Weber — Not consistently referred for BC or ABC.

Monochord			
	AC	15,000 d.v.	15,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The air insufflation has not made any change in the hearing tests. This patient's answers during the Weber test were most unreliable. This might have been a language difficulty.

CASE No. 11 — Mr. R. F., age 21. Admitted March 29, 1936.

DIAGNOSIS: Not yet determined. Posterior fossa tumor suspect (not confirmed).

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 30, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Otorrhea in childhood; side not known. *Right drum* — thickened in posterior half. *Left drum* — shows narrow band of thickening concentric with periphery in the posterior inferior quadrant.

HEARING EXAMINATION

AUDIOGRAM — (Similar to Fig. 10) — Subclass, Right A_1 C_6 down to 50 s.u.; Left A_1.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	35 seconds	35 seconds
	ABC	45 seconds	45 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: April 2, 1936. Ventriculography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 3, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — (Similar to Fig. 10) — Subclass, Right A_1 C_6 down to 50 s.u.; Left A_1.

(Masking left ear with air caused further drop of 20 s.u. at 4096 and 8192 d.v. in right ear.)

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	45 s.u.	45 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	35 seconds	35 seconds
	ABC	50 seconds	50 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Ventriculography caused no change in hearing. The dip for the high notes of the audiometer in the right ear may possibly be explained by the history of the old middle ear disease. On the other hand, it may have been caused by an undiagnosed intracranial lesion. An exploratory suboccipital myoplastic craniotomy April 7, 1936, after the last hearing examination did not reveal any evidence of a neoplasm.

CASE NO. 12 — Mr. R. S., age 46. Admitted March 23, 1936.

DIAGNOSIS: Cerebral thrombosis, right middle cerebral artery. Internal hydrocephalus, cause undetermined.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 25, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	25 seconds	20 seconds
	ABC	35 seconds	35 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	18,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE: April 1, 1936. Encephalography.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 2, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 each ear.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	55 s.u.	55 s.u.

c^1 (256)	BC	75 s.u.	75 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests

c^2 (512)	AC	70 seconds	70 seconds
	BC	22 seconds	22 seconds
	ABC	35 seconds	35 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Encephalography has not caused any alteration of hearing.

CASE No. 13 — Mr. A. W., age 38. Admitted February 17, 1936.

DIAGNOSIS: Multiple fractures of the skull with left peripheral facial paralysis.

NOTE: This injury occurred November, 1935. There was bleeding from both ears.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 20, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Some evidence of injury to both drums.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right B_1; Left A_2.

BONE CONDUCTION TESTS

Tuning Fork Tests

	RIGHT	*LEFT*
Rinné	Negative	Negative
Schwabach	Normal	Normal
Weber — Not referred.		
Whispered Voice	8 feet	6 feet
Normal Voice	25 feet	25 feet

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

NOTE: Above tests not done by present investigators.

OPERATIVE PROCEDURE:

1. February 26, 1936. Spinal subarachnoid insufflation.
2. March 7, 1936. Bilateral trephination and subdural insufflation.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 9, 1936

NOSE and THROAT — Normal.

OTOSCOPY — As previously reported.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right B_1; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.

c^1 (256)	BC	55 s.u.	60 s.u.
	ABC	45 s.u.	40 s.u.

Tuning Fork Tests

c^2 (512)	AC	60 seconds	60 seconds
	BC	20 seconds	25 seconds
	ABC	40 seconds	45 seconds

Weber — Not definitely referred for BC or ABC.

Monochord	AC	13,000 d.v.	14,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

NOTE: There has been a slight improvement in hearing since February 20, 1936 (eighteen days), which may be due to healing of the middle ear condition, since the previous examination, or it may be due to variation in the examination methods. The slight loss of hearing probably due to a middle ear lesion is further manifested by a shortening of the interval between BC and ABC in the audiometer.

IMPRESSION: Encephalography has not made any essential change in the hearing tests.

SUMMARY OF GROUP ONE

There was no appreciable change in hearing noted in any of these patients examined within twenty-four hours following encephalography or ventriculography. Some of the patients did have hearing losses before the procedure but these were unchanged. One must conclude, therefore, that in 13 patients these procedures which presumably cause changes in intracranial pressure have not caused any effect upon the hearing.

An approach to the problem with accurate measurements of pressure changes would be of great value, or an examination made before and after the dura had been opened without brain injury, as for a simple decompression, would be more definite but such opportunities are rare. The difficulty of making a more definite post-operative check upon this conclusion when dealing with patients is very great. Once the dura is opened and brain tissue is removed, it is no longer possible to say what changes in hearing, if any, are due to change of intracranial pressure as distinct from any effect that removal of the brain tissue might have.

GROUP TWO

Cerebral Tissue Removal

This group comprises fourteen patients all of whom had an intracranial operation for the removal of cerebral tissue. Twelve of the patients had epilepsy and the operations were to remove

cerebral cicatrices. In two patients, the brain removal followed a recent skull fracture with brain damage. The amount of cortical and subcortical brain removal varied in each case, but an approximate estimate is given in each report. Five of the patients showed some hearing loss.

CASE REPORTS

Case 1 is a member of this group because cerebral tissue was removed. The complete case report is included in Group One because the hearing was also tested before and after encephalography.

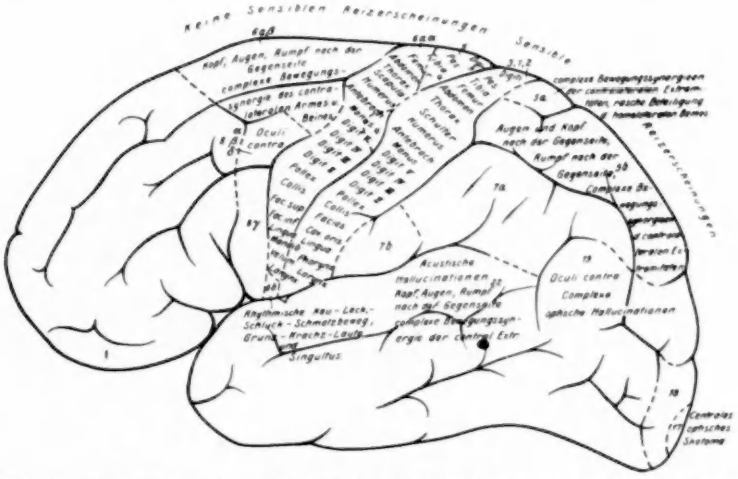


FIG. 5 — (McNally) — Diagram of the human cortex after Foerster and Penfield (Penfield and Gage 1933).

CASE NO. 14 — Miss F. F., age 19. Last admission August 28, 1935.

DIAGNOSIS: Epilepsy, Jacksonian (motor), right. Multiple cerebral cicatrices, right hemisphere.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION
August, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Right drum, slightly retracted. Left drum, normal.

HEARING EXAMINATION — Normal to superficial examination.

OPERATIVE PROCEDURES:

November, 1932 — Encephalography.
October, 1934 — Encephalography.
November 6, 1935 — Operator, W. G. Penfield. Myoplastic subtemporal craniotomy and separation of adhesions, left.

March 20, 1935 — Operator, W. G. Penfield. Right parietal osteoplastic craniotomy, and removal of cerebral cicatrix.

September, 1935 — Operator, W. G. Penfield. Right parietal osteoplastic craniotomy, and removal of cerebral cicatrix.

Cicatrix removal, March 20, 1935, in size, 1 cm. deep x 3 x 4 cm., so that the whole tip of the temporal lobe was removed to a depth of 1 cm.

Cicatrix removal, September 11, 1935, which was in the right post-central gyrus anteriorly (area 7b, Fig. 5), in size 3x3x2 cm., and weighing 7 grams.

December 16, 1935 — Patient unimproved, still having attacks.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 16, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Right drum, slightly retracted. Left drum, normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, right and left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	30 seconds	30 seconds
Weber — Not lateralized for BC or ABC.			
Monochord	AC	18,000 d.v.	19,000 d.v.
	BC	19,000 d.v.	20,000 d.v.
	ABC	21,000 d.v.	21,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

IMPRESSION: Multiple intracranial operative procedures have not caused any significant loss of hearing.

CASE NO. 15 — Mr. G. G., age 19. Last admission Nov. 12, 1931.

DIAGNOSIS: Epilepsy and meningo-cerebral cicatrix of the right frontal lobe.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

October 27, 1931

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

November 13, 1931 — Operator, W. G. Penfield. Right fronto-parietal craniotomy, and amputation of the right frontal lobe. The right frontal lobe was cystic and was amputated, including the tip of the lateral ventricle. The tissue over an area of 4x4x4 cm. deep,

weighing 34 grams, was removed. It is noted that there is some suggestion of a lesion in the left frontal pole, but this area was not removed.

December 16, 1935 — The patient is in good general health and has had no return of the seizures.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 16, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, right and left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	60 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	85 seconds	85 seconds
	BC	30 seconds	30 seconds
Weber — Not lateralized.			
Monochord	AC	17,000 d.v.	18,000 d.v.
	BC	18,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

IMPRESSION: The removal of the right frontal lobe has not caused any loss of hearing.

CASE No. 16 — Miss I. H., age 15. Admitted June 24, 1932.

DIAGNOSIS: Traumatic meningo-porencephaly. Multiple cysts of left frontal lobe. Cranial defect. Jacksonian Epilepsy.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

June 24, 1932

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal to superficial examination.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

NOTE: December 5, 1932. Patient had grippe, and otorrhea right and left.

OPERATIVE PROCEDURES:

1. June, 1918 — Aspiration of fluid from cyst of left frontal eminence.
2. November, 1919 — Ventriculography.
3. June 25, 1932 — Operator, W. V. Cone. Left frontal exploratory craniotomy, removal of cerebral cicatrix, and plastic repair of skull defect. A depressed bone fracture was removed leaving a bone defect in the skull of 8 cm. in diameter. All the lateral portion of the left frontal lobe was removed over an area of 7 cm., together with the enlarged anterior horn of the left ventricle. A celloidin plate was inserted in the bone defect.
4. December 20, 1932 — Operator, W. V. Cone. Plastic repair of cranial defect.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 19, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Drums normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left A_2.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	60 s.u.	50 s.u.
	ABC	50 s.u.	55 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	30 seconds	30 seconds
	ABC	40 seconds	40 seconds
Weber — Not recorded.			
Monochord	AC	19,000 d.v.	19,000 d.v.
	BC	20,000 d.v.	20,000 d.v.

NOTE: Patient a very poor witness.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

IMPRESSION: The removal of the left frontal lobe has not caused any definite loss of hearing. The fact that the patient was such a poor witness may be in some way due to her brain lesion.

CASE NO. 17 — Mr. D. K., age 23. Admitted January 4, 1936.

DIAGNOSIS: Extensive depressed fracture, left frontal region and base of the skull. Laceration of the dura, and left frontal lobe.

NOTE: Ophthalmoplegia of the right eye except for the external rectus.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

January 4, 1936

NOSE and THROAT — Both nares contained crusted blood, but passages not obstructed.

OTOSCOPY — Both canals filled with old and recent blood. Fresh bleeding in each ear from the wall of the canal near the drum; both drums dusky purple in color, appearing to have blood behind them.

HEARING EXAMINATION — Not done. Patient stuporous.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURE:

January 4, 1936 — Operator, W. V. Cone. Removal of depressed fracture of the skull; débridement of the left frontal lobe; removal of mucous membrane of frontal sinuses. Suture of lacerated wound of the dura. Brain tissue filled the frontal sinuses, the comminution of the left frontal region and base was most extensive, and the roof of the orbit was in many pieces. Many comminuted fragments as far back as the sphenoid. Tip of frontal lobe entirely destroyed to a depth of 3 cm.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 2, 1936

NOSE and THROAT — Thickened exudate more or less crusted on the posterior wall of the nasopharynx. Otherwise negative.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 6).

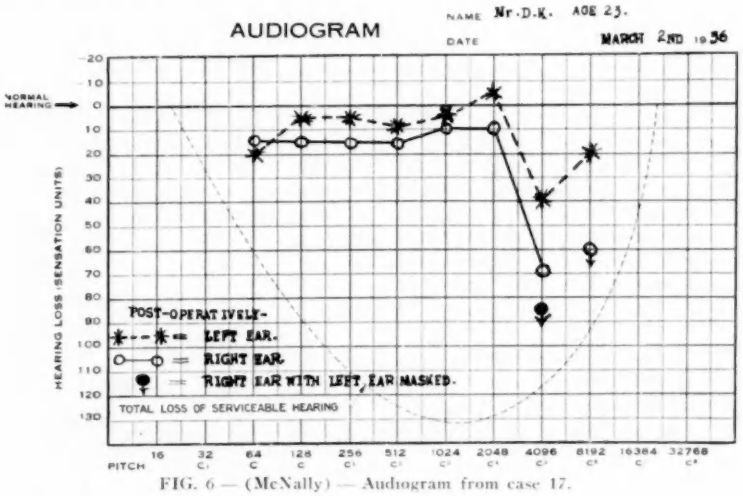


FIG. 6 — (McNally) — Audiogram from case 17.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	55 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
c^1 (256)	BC	55 s.u.	45 s.u.
	ABC	35 s.u.	35 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	70 seconds
	BC	30 seconds	30 seconds
	ABC	45 seconds	45 seconds

Weber — Referred to the right ear by BC. Not referred for ABC.

Monochord	AC	9,000 d.v.	16,000 d.v.
	BC	16,000 d.v.	19,000 d.v.

VESTIBULAR EXAMINATION

Cold Caloric Test — Stimulation of the ho.izontal and vertical canals of each ear elicits a normal nystagmus from the left eye and an occasional atypical nystagmoid movement of the right eye.

Rotation Test — (Head to one side so that the turning is about a bi-temporal axis) — Normal response from the left eye, and an atypical response from the right eye, the vertical canals being stimulated.

IMPRESSION: Hearing for low tones and for bone conduction normal in both ears. The audiogram shows a high tone loss, marked on the right side, which was partly confirmed by the monochord. This would suggest partial nerve deafness on the right side. Both vestibules are reacting nor-

mally. At the time of the injury, there was bleeding from both ears suggesting bilateral middle ear trauma, which might be sufficient to account for the hearing lesion noted. The normal vestibular reactions are opposed to a traumatic internal ear lesion at the time of the fracture. There was evidently extensive trauma in the left frontal region, and the right-sided ophthalmoplegia would suggest extensive trauma in the right frontal basal region. It is impossible to say whether these frontal lesions or the middle ear lesions at the time of the accident are responsible for the hearing loss.

Case No. 18 — Mr. T. L., age 41. Admitted August 2, 1930.

Diagnosis: Depressed fracture of the skull, right temporal region. Laceration of the dura with softening of the brain. Intracerebral hemorrhage.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

August 2, 1930

(Examination could not be completed because patient unconscious.)

NOSE and THROAT — Not examined.

OTOSCOPY — Blood clot in right canal (not disturbed).

HEARING EXAMINATION — Not tested.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

Note: At the time of the accident there was profuse bleeding from the nose and right ear.

Operative Procedures: August 4, 1930. Operator, Dr. W. V. Cone.

1. Elevation of depressed fracture of the skull and removal of bony fragments.
2. Subtemporal decompression, right. Aspiration of damaged brain. Removal of two intracerebral blood clots. The softened brain involved almost the entire right temporal lobe.

March, 1930 — General condition is very satisfactory.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

September 7, 1930

NOSE and THROAT — Not examined.

OTOSCOPY

Right Drum — Blood clot in the right canal. After removal the right drum was slightly dull and thickened but apparently intact.

Left Drum — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3 C_4 down to 45 s.u. (somewhat similar to Fig. 18). Left A_2.

BONE CONDUCTION TESTS

Tuning Fork Tests c^2 (512)

	RIGHT	*LEFT*
Rinné	Positive	Positive

Weber — Not lateralized.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

Note: Examination not done during this investigation.

IMPRESSION: The slight hearing loss in the right ear is characterized by a dip at 8192 d.v. to 45 sensation units. Unfortunately this test did not include the monochord. The slight hearing loss in the right ear may be due to ear damage at the time of the accident or it may be due to the intracranial lesion.

CASE No. 19 — Mr. M. McK., age 21. Admitted June 12, 1934.

DIAGNOSIS: Jacksonian Epilepsy. Cicatrix, following left frontal lobe abscess.

NOTE: In August 1931, following an acute left frontal sinus abscess and osteomyelitis, the patient developed a left frontal lobe abscess, which was drained through the frontal sinus. The following year he began having definite Jacksonian attacks.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

June 12, 1934

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal to superficial examination.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

June 13, 1934 — Operator, W. V. Cone. Left frontal osteoplastic craniotomy, and amputation of the left frontal lobe. The left frontal lobe was completely removed back to the beginning of the corpus callosum at the midline, and back to the tip of the left lateral ventricle. The size of the specimen removed was 7x4x5 cm., and weighed 44 grams.

December 12, 1935 — No recurrence of seizures and the patient is in good general health. He is able to carry on his medical studies.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 12, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, right and left.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	75 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.
Tuning Fork Tests c^2 (512)			
Rinné		AC greater than BC	AC greater than BC
Schwabach — Normal.			
Weber — Not lateralized.			
Monochord	AC	19,000 d.v.	19,000 d.v.
	BC	20,000 d.v.	23,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Tests* — Not done.

IMPRESSION: Removal of the left frontal lobe has not caused any significant loss of hearing.

CASE No. 20 — Mr. F. R., age 20. Admitted October 22, 1935.

DIAGNOSIS: Epilepsy, Jacksonian, motor. Cerebral cicatrix left frontal lobe.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

October 22, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — No note.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

October 25, 1935 — Encephalography.

October 30, 1935 — Operator, W. G. Penfield. Left osteoplastic craniotomy and removal of cerebral cicatrix. A section 3 cm. anterior to the precentral gyrus was removed, in size 9x4.5x3.5 cm., and weighing 87.3 grams. It extended down to the ventricle.

February 20, 1936 — Improved but not free from attacks.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

February 20, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — (Similar to Fig. 9) — Subclass, Right A_1 with dip at 4096 to 50 s.u.; Left A_1 with dip at 4096 to 50 s.u.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	75 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	65 s.u.	65 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	80 seconds	80 seconds
	BC	30 seconds	30 seconds
	ABC	50 seconds	50 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Rotation Test* — Normal.

IMPRESSION: Removal of left frontal lobe has not produced any significant loss of hearing except for a bilateral dip in the audiogram at 4096 d.v. This case will be discussed with other cases showing a similar audiogram.

CASE No. 21 — Mr. D. R., age 18. Admitted June, 1932. Encephalogram. Admitted May 2, 1935.

DIAGNOSIS:

1. Frontal atrophy of the brain, left temporal region, post-traumatic.
2. Epilepsy, Jacksonian, motor. (Epilepsy, focal cortical seizures.)

NOTE: In October 1923, the patient was knocked down by a car. He was taken to the hospital, where a bilateral subtemporal decompression was done, and a subdural hematoma found on the right side. Attacks of epilepsy began within 6 months of the accident.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

May 2, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

June 13, 1932 — Encephalography.

May 14, 1935 — Operator, W. G. Penfield. Left parieto-temporal osteoplastic craniotomy and removal of cerebral atrophy. A localized area of atrophy was removed from the posterior part of the superior temporal convolution measuring 1x1.5x1.5 cm.

April 7, 1936 — The patient is well, no attacks, continues to take luminal.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 7, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 with peak 4096, Right and Left to — 10 s.u.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	22 seconds	25 seconds
	ABC	50 seconds	50 seconds

Weber — Not lateralized for BC or ABC.

Monochord	AC	17,000 d.v.	16,000 d.v.
	BC	18,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Removal of brain tissue from the left parieto-temporal region has not produced any abnormality of hearing.

CASE No. 22 — Mr. F. S., age 38. Admitted April 28, 1935.

DIAGNOSIS: Right frontal meningocerebral cicatrix. Epilepsy, Jacksonian.

NOTE: Seizures for 6 years. No history of trauma was obtained.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

April 18, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.

OPERATIVE PROCEDURES:

April 23, 1935 — Operator, W. G. Penfield. Right parietal osteoplastic craniotomy and excision of cerebral cicatrix. An area of brain situated immediately in front of the precentral gyrus and extending down to the falx was excised. Measurements 5x3x4 cm.

March 17, 1936 — Patient well, and has had no major seizures but recently has had a return of epigastric aura.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 9, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3 C_6 down to 55 s.u. (somewhat similar to Fig. 18).
Left A_3 C_4 down to 60 s.u. (somewhat similar to Fig. 10).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	75 s.u.
	ABC	60 s.u.	60 s.u.
c^1 (256)	BC	65 s.u.	75 s.u.
	ABC	50 s.u.	55 s.u.

Weber — Not lateralized for BC or ABC.

Tuning Fork Tests			
c^2 (512)	AC	55 seconds	55 seconds
	BC	20 seconds	15 seconds
	ABC	30 seconds	30 seconds
Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR REACTIONS — *Cold Caloric Test* — Normal.

IMPRESSION: The abrupt high tone loss in the audiogram and the shortening of the times for air conduction of the fork and slight lowering of the monochord are the three definite departures from normal, shown post-operatively in this case. Such a high tone loss is frequently associated with cochlear damage (nerve deafness).

CASE No. 23 — Mr. W. S., age 27. Admitted February 11, 1931.

DIAGNOSIS: Epilepsy, Jacksonian. Cerebral cicatrix and atrophy in the left parieto-occipital area.

NOTE: Head injury at the age of 9 years, and attacks since the age of 11.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 11, 1931

NOSE and THROAT — Not examined.

OTOSCOPY — No examination.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.

OPERATIVE PROCEDURES:

February 13, 1931 — Encephalography.

February 20, 1931 — Operator, Dr. Wilder Penfield. Left parietal osteoplastic craniotomy with removal of cerebral cicatrix. There was a moderate amount of atrophy of the whole left hemisphere, and a very deep fissure of Sylvius. At the upper and posterior end of this (left parieto-occipital area), one convolution was so atrophied that it was only about 2 mm. wide. A surrounding area of about 8 cm. diameter showed less severe atrophy and was situated directly behind the motor gyrus on a level with the arm area. The extent of the tissue excised was about 4x4x4 cm., and its weight about 15 grams. It extended down into the ventricle, about 2 or 3 cm. of its roof was excised.

December, 1935 — Patient well. No seizures.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 19, 1935

NOSE and THROAT — Not examined.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right E_1; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	80 s.u.
	ABC	55 s.u.	50 s.u.

Tuning Fork Tests			
c^2 (512)	AC	90 seconds	90 seconds
	BC	25 seconds	20 seconds

Weber — Not lateralized.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — Not done.

IMPRESSION: The removal of the cerebral cicatrix from the parieto-occipital area has not caused any significant hearing loss.

CASE No. 24 — Miss M. S., age 22. Admitted February 17, 1933.

DIAGNOSIS: Jacksonian epilepsy. Meningo-cerebral cicatrix, occipito-parietal region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION
February 17, 1933

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.

OPERATIVE PROCEDURE:

February 23, 1933 — Operator, Dr. Wilder Penfield. Right, occipito-parietal osteoplastic craniotomy, with removal of cerebral cicatrix of the occipito-parietal region, about 7x5 cm. in area and completely unroofing the lateral ventricle over an area of 4x2 cm.

Note: December 12, 1935 — Patient well and has had no major attacks. However, she has had some minor sensory phenomena.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION
December 12, 1935

NOSE and THROAT — Not examined.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right B_1; Left B_1.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	70 s.u.	75 s.u.
	ABC	60 s.u.	55 s.u.
Tuning Fork Tests c^2 (512)			
Rinné		AC greater than BC	AC greater than BC
Schwabach		Normal	Normal
Weber — Not lateralized for BC or ABC.			
Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION — Not done.

IMPRESSION: The hearing apparently was not affected by the lesion in the occipito-parietal region.

CASE No. 25 — Miss J. R., age 13. Admitted April 24, 1933.

DIAGNOSIS: Jacksonian Epilepsy. Cerebral cicatrix in small precentral gyrus, right, near the midline.

NOTE: No history of trauma.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION
April 24, 1933

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURE:

May 2, 1933 — Operator, W. G. Penfield. Right osteoplastic craniotomy and excision of cerebral cicatrix. A small, hard, gyrus, just anterior to the precentral gyrus near the midline, was removed, with some of the gyrus in front and behind. The excision was not carried into the ventricle.

December 19, 1935 — No further seizures, and the patient is in excellent general health.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

December 19, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, right and left.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	65 s.u.	70 s.u.
	ABC	55 s.u.	55 s.u.
Tuning Fork Tests			
c^2 (512)	AC	85 seconds	85 seconds
	BC	30 seconds	35 seconds
Weber — Not recorded.			
Monochord	AC	19,000 d.v.	21,000 d.v.
	BC	20,000 d.v.	22,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

IMPRESSION: The removal of a small area of brain tissue from the right frontal lobe has not caused any loss of hearing.

CASE No. 26 — Mr. F. W., age 17. Last admission April, 1936.

DIAGNOSIS: Epilepsy, combined form, grand and petit mal, idiopathic.
NOTE: Attacks since August 1934. History of trauma at the age of 11 years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

June, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.
NOTE: Superficial hearing examinations were done on the previous admissions and were normal.

OPERATIVE PROCEDURES:

October 3, 1934 — Encephalography.

October 6, 1934 — Encephalography.

March 15, 1935 — Encephalography.

June 1, 1935 — Operator, Dr. Wilder Penfield. Left parietal craniotomy and excision of leptomeningeal cicatrix. Removal of cicatrix from the left frontal adversive field near the Pacchionian body. The cicatrix measured 1x1x2 cm.

April 7, 1936 — Unimproved. Attacks continue.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 7, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2 C_4 down to 55 s.u., somewhat similar to Fig. 18; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	65 s.u.
	ABC	55 s.u.	55 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	70 seconds
	BC	25 seconds	30 seconds
	ABC	40 seconds	45 seconds

Weber — Tendency to lateralize BC to the left when the fork was placed in the midline, but it was not lateralized for ABC.

Monochord	AC	11,000 d.v.	16,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The audiometer and the monochord show a high tone loss by air conduction in the right ear. It is unlikely that such a small lesion in the frontal lobe would cause such a hearing loss when much more extensive lesions have had no effect upon the hearing.

SUMMARY OF GROUP TWO

In this group of fourteen patients, the removal of cerebral tissue was from the following regions:

Frontal Lobe

Right ..... Cases 15, 22, 25

Left ..... Cases 16, 17, 19, 20, 26

Parietal Lobe

Right ..... Case 24

Left ..... Cases 1, 21, 23

Temporal Lobe — Right ..... Cases 14, 18

The hearing was normal in nine of the patients. In two of the five patients showing slight departures from normal hearing, there had been bleeding from the ears following skull fracture. Considerable evidence is accumulating to show that a middle ear lesion may be accompanied by a loss of high tones (see Ciocco, 1932, and Gottlieb, 1933).

In the three remaining patients it is difficult to ascribe the high tone hearing loss to the brain lesion because the lesions in cases 16 and 19, in which the hearing was normal, were apparently just as large, and also from the left frontal lobe area, as in cases 20 and 26, which showed a hearing loss. The lesion in case 25, in which the hearing was normal, was anterior to the precentral gyrus, just as in case 22, which showed a hearing loss. Furthermore, the two patients of group two with temporal lobe lesions had normal hearing, yet these lobes are thought to be more concerned with hearing than are the frontal lobes.

The five patients with hearing loss are later discussed in detail under the following headings:

Case 17 — Abrupt high tone loss
Cases 18, 22, 26 — Gradual high tone loss
Case 20 — Audiogram dip at 4096 d.v.

GROUP THREE

Cerebral Tumors

There are nine patients in this group, each of whom had a tumor removed from the cerebrum. Seven of the patients showed some hearing loss post-operatively. Unfortunately, there were no detailed pre-operative hearing tests carried out on the patients of this group, except in Case 32, and even this examination was not done during the present investigation.

CASE REPORTS

CASE NO. 27 — Mrs. G. B., age 35. Admitted May 27, 1933.

DIAGNOSIS: Meningeal fibroblastoma of the falx cerebri, left frontal lobe.

NOTE: Severe bilateral tinnitus for 5 years. Vertigo for 5 months.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION
May 27, 1933

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURE:

June 1, 1933 — Operator, Dr. W. V. Cone. Left osteoplastic craniotomy and removal of tumor which was the size of a tennis ball, and weighed approximately 120 grams. It arose from the falx and invaded the left anterior fossa, displacing the left frontal lobe. The tumor was removed completely, together with the overlying frontal lobe tissue.

Note: April 30, 1936 — The patient was well and in good general health.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 30, 1936

NOSE and THROAT — Normal

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	70 s.u.
	ABC	50 s.u.	55 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	50 s.u.	45 s.u.

Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	35 seconds	35 seconds
	ABC	45 seconds	45 seconds

Weber — Not lateralized. Blocking either ear caused the sound to be referred to that ear.

Monochord			
	AC	14,000 d.v.	14,000 d.v.
	BC	16,000 d.v.	15,000 d.v.

IMPRESSION: The frontal lobe lesion has not caused any change in hearing.

CASE NO. 28 — Miss H. B., age 24. Admitted August 27, 1935.

DIAGNOSIS: Meningeal fibroblastoma of the right frontal region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

September 12, 1935

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Normal.

Left Drum — Small atrophic area in the postero-inferior region. Otorrhea, left, for 2 years in infancy.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.

OPERATIVE PROCEDURES:

August 30, 1935 — Ventriculography.

August 30, 1935 — Operator, Dr. Wilder Penfield. Right parietal osteoplastic craniotomy and removal of right frontal tumor. It weighed 62.3 grams and had a dural attachment of 6.0x3.5 cm. The hemisphere was pushed out of the way, but was not otherwise involved by the tumor. Almost no brain tissue was removed at the operation.

Note: April 6, 1936 — Patient in good health but began having epileptic seizures during the month of April, 1936.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 6, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	65 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	45 s.u.	45 s.u.

Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	40 seconds	40 seconds
	ABC	60 seconds	60 seconds

Weber — Not lateralized for BC or ABC.

Monochord			
	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: After the removal of the tumor from the frontal lobe the hearing was essentially normal.

CASE No. 29 — Mr. C. C., age 36. Admitted February 21, 1936.

DIAGNOSIS: Meningeal fibroblastoma invading the post-frontal region of the skull.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 21, 1936

NOSE and THROAT — Some evidence of chronic rhinitis.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Not done.

OPERATIVE PROCEDURES:

February 29, 1936 — Operator, Dr. W. V. Cone. Bilateral frontal craniotomy with removal of meningeal fibroblastoma, infiltrating the

skull and pericranium and arising from the dura and presenting on both sides of the median longitudinal sinus. The tumor mass covered an area of 6.7x6.5x4.0 cm., and weighed 95 grams. It arose from the undersurface of the dura on both sides of the falx cerebrum and occluded the median longitudinal sinus over a distance of 10 cm.

February 29, 1936 — Operator, Dr. D. L. Reeves. Excision of fascia lata from left thigh for transplant.

April 23, 1936 — Patient working and in good general health.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 23, 1936

NOSE and THROAT — Pus and polypi in the right nostril. In 1927 operative treatment of the sinuses left side. Chronic tonsillitis.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3 with 4096 dip to 30 s.u. (similar to Fig. 9). Left, A_3 with 4096 dip to 30 s.u. (similar to Fig. 9).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	60 s.u.	60 s.u.
	ABC	50 s.u.	45 s.u.
c^1 (256)	BC	65 s.u.	70 s.u.
	ABC	50 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	25 seconds	25 seconds
	ABC	35 seconds	35 seconds
Weber — Not referred for BC or ABC.			
Monochord	AC	14,000 d.v.	14,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The chronic rhinitis with possible eustachian tube change in this patient may be a factor in explaining slight hearing loss.

CASE NO. 30 — Mr. W. L., age 54. Admitted April 22, 1936.

DIAGNOSIS: Glioblastoma multiforme, left occipito-parietal region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

April 22, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

April 23, 1936 — Operator, A. R. Elvidge. Ventriculography.

April 23, 1936 — Operator, A. R. Elvidge. Left occipito-parietal osteoplastic craniotomy and removal of tumor. At completion of the left occipital lobectomy, tissue weighing 277.5 grams had been removed.

May 14, 1936 — Post-operative recovery continues satisfactorily. The hearing tests were done on the 13th day post-operatively, but the patients mental condition was quite clear.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 6, 1936

NOSE and THROAT — Nose, normal. Chronic tonsillitis.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right D_1; Left B_1.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	75 s.u.
	ABC	60 s.u.	55 s.u.
c^1 (256)	BC	65 s.u.	70 s.u.
	ABC	50 s.u.	55 s.u.

Tuning Fork Tests			
c^2 (512)	AC	60 seconds	65 seconds
	BC	20 seconds	25 seconds
	ABC	30 seconds	35 seconds

Weber — Not lateralized for BC or ABC.

Monochord			
	AC	13,000 d.v.	13,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The slight loss of hearing shown in the audiogram is consistent with the patient's age, and the monochord is within the lower limits of normal. This patient could be considered within the group of normals. It is just possible, if he were tested at a later date after complete recovery from his operation, that any slight loss of hearing shown at this time might have disappeared. Removal of part of the left occipital lobe cannot be said to have had any significant effect upon the hearing.

CASE No. 31 — Mr. A. L., age 43. Admitted October 8, 1935.

DIAGNOSIS: Meningeal fibroblastoma, right parietal region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

October 8, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination; says hearing has been impaired in the left ear since the age of 24 years.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

1. October 12, 1935 — Encephalography.
2. October 12, 1935 — Operator, W. V. Cone. Right parietal craniotomy with partial removal of meningeal fibroblastoma attached to the falx and invading the median longitudinal sinus.
3. October 21, 1935 — Operator, W. V. Cone. Re-opening of scalp flap and complete removal of the tumor. Fascial transplant from the thigh to close the dura.

   The large meningeal fibroblastoma was removed in two stages. It obliterated the median longitudinal sinus, and was infiltrating the falx. Because of the skull involvement, the bone removal had to be extensive. Tumor weighing 124 grams was removed at the first operation, which included all that part of the tumor which extended into the right hemisphere and most of it which was attached to the falx cerebri. At the second operation, the falx of the cerebrum and the median longitudinal sinus were resected.

   April 21, 1936 — Patient continues to improve.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 21, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3; Left B_1.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	75 s.u.
	ABC	65 s.u.	65 s.u.
c^1 (256)	BC	60 s.u.	55 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	60 seconds	35 seconds
	BC	18 seconds	23 seconds
	ABC	27 seconds	28 seconds

Weber — Referred to the left when placed to the right of the midline except in the right mastoid region. Not referred for ABC.

Monochord	AC	16,000 d.v.	15,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The slight drop shown by the audiogram for the upper tones on the left side is in keeping with the patient's idea of hearing loss in the left ear over a period of years. The slight hearing loss is of the middle ear deafness type, there being no evidence of any nerve involvement. The rather sharp drop in the left ear for A.C. for the fork c^2 (512) is not in keeping with any of the other findings, except that it probably does explain why the Weber is referred to the left, this A.C. drop probably acting in the same way as would blocking the left ear. In the absense of a complete pre-operative examination of hearing, there is nothing to show that these slight hearing losses are due to the cerebral lesion.

CASE No. 32 — Mr. H. M., age 33. Admitted April 1, 1935.

DIAGNOSIS: Oligodendroglioma, right frontal lobe. Epilepsy, focal cortical seizures (Jacksonian) motor. Bilateral chronic suppurative otitis media, quiescent.

NOTE: Intermittent discharge from the right ear since childhood, recently has been dry; left ear has discharged for the past five years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 15, 1935

NOSE and THROAT — Chronic septic tonsillitis and chronic rhinopharyngitis.

OTOSCOPY

Right Drum — Much scarred and quite dry.
Left Drum — Foul-smelling pus. Large anterior perforation with granulations.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 7).

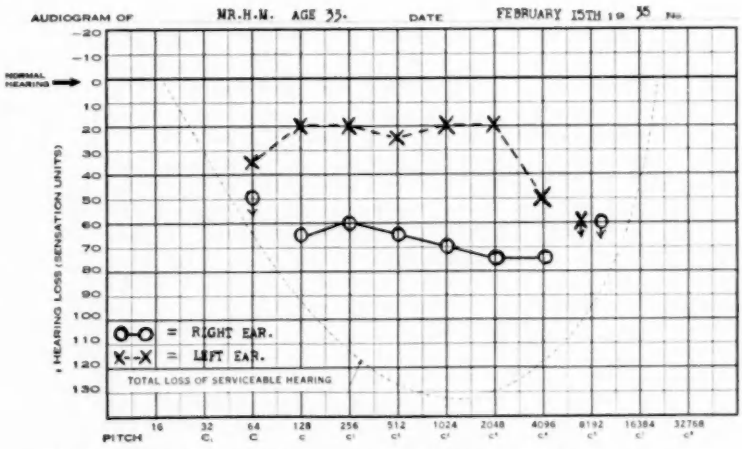


FIG. 7 — (McNally) — Audiogram from case 32 (Pre-operative examination).

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	ABC	65 s.u.	60 s.u.
Tuning Fork Tests c^2 (512)			
Rinné		BC greater than AC	AC greater than BC
Schwabach		Normal	+ 15 seconds
Watch Test		8/30" 0/30"	8/30"
Whispered Voice		Not heard	3 feet
Conversational Voice		12 feet	12 feet
Monochord	AC	7,000 d.v.	12,000 d.v.

VESTIBULAR EXAMINATION

Rotation Test — Normal.
Cold Caloric Test — Not done.

NOTE: The above tests not done by present investigators.

OPERATIVE PROCEDURES:

February 18, 1935 — Operator, Keith Hutchinson. Radical mastoidectomy, left.

April 6, 1935 — Operator, W. G. Penfield. Right osteoplastic craniotomy with removal of tumor from the right precentral gyrus whose surface measurement was 5x3 cm. and weight 19 grams.

February 27, 1936 — No seizures. General condition is very good.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

February 27, 1936

(These tests were repeated on two successive days)

NOSE and THROAT — Chronic septic tonsillitis. Irregular septal deviation. Chronic rhinitis.

OTOSCOPY

Right Drum — Intact, thickened and scarred, suggesting an old middle ear condition, quiescent.

Left Ear — Healed radical mastoid cavity.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 8).

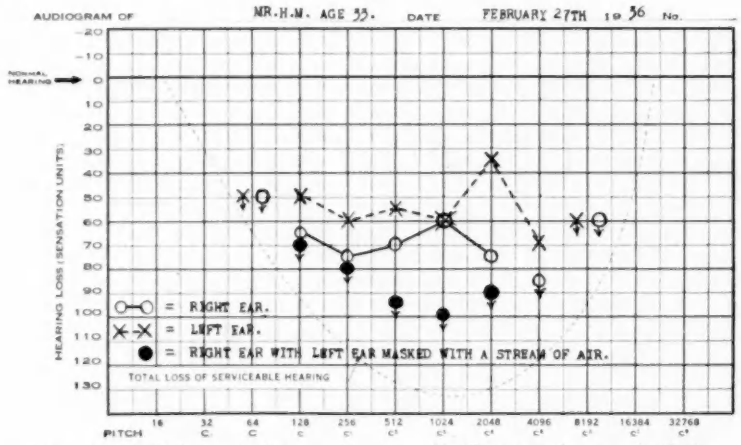


FIG. 8 — (McNally) — Audiogram from case 32 (Post-operative examination).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with A.M.	No masking	Op. ear masked with A.M.
c^2 (512)	BC	75 s.u.	N.H.	60 s.u.	60 s.u.
	ABC	65 s.u.	N.H.	60 s.u.	60 s.u.
c^1 (256)	BC	63 s.u.	N.H.	55 s.u.	55 s.u.
	ABC	60 s.u.	N.H.	55 s.u.	55 s.u.
Tuning Fork Tests					
c^2 (512)	AC	5 seconds	N.H.	15 seconds	10 seconds
	BC	25 seconds	N.H.	25 seconds	25 seconds
	ABC	27 seconds	N.H.	20 seconds	27 seconds

Weber — From the midline of the vertex and from any point to the right of this, the sound is referred to the right ear. To the left of the midline, the sound is referred to the left. Masking the right ear with a current of air had no consistent effect. Masking the left ear with a current of air eliminates all hearing for the fork.

		RIGHT		LEFT	
		No masking	Op. ear masked with A.M.	No masking	Op. ear masked with A.M.
Monochord	AC	N.H.		10,000 d.v.	
	BC	14,000 d.v.	6,000 d.v.	15,000 d.v.	15,000 d.v.
	ABC	14,000 d.v.		15,000 d.v.	
Watch Test		0/6″		1/4/6″	
Whispered Voice		N.H.		N.H.	
Conversational Voice		N.H.		2 feet	

VESTIBULAR EXAMINATION

Cold Caloric Test — Right, no response to ethyl chloride; Left, normal response.
Rotation Test — Symmetrical, but only slight response after turning to either side with the head erect.

IMPRESSION: The hearing test before the radical mastoid operation was not accompanied by masking. It is possible that the hearing shown for the right ear could have been completely eliminated by masking the left ear. Before the radical mastoid operation there was apparently fairly good hearing in the left ear, and a reacting labyrinth. Following the mastoid and intracranial operation, there has been a marked reduction in the hearing of the left ear, and a complete loss of reaction from the left vestibule. It is interesting to note that although there is very little if any hearing in the right ear, the vestibular reactions on the right side are still within normal limits, whereas in the left or hearing ear, the vestibular reactions have disappeared. This is very difficult to explain on the basis of the radical mastoid operation, because even though there has been a decrease in hearing of the left ear, if one presumed there had been a post-operative labyrinthitis sufficiently severe to destroy the vestibular reactions, the hearing should have been destroyed rather than simply reduced. On the other hand, it is not in keeping with our observations of other cases that have had similar small cortical defects in the region of the motor area that they should have such profound effects upon the vestibular apparatus. This case illustrates very well the value of masking even in the presence of two more or less severely affected ears. This is another instance of the Weber test being most misleading, since it tended to be referred to the more deaf ear. It is interesting to note that although air conduction for the Monochord was much reduced in both ears (completely eliminated on the right side), for bone conduction the range was very good in both ears, and masking the left ear did not completely eliminate the hearing for bone conduction in the right ear. This may be another instance of the difficulty of masking out the Monochord. This is more likely than that it should be an indication of some residual hearing in the right ear. It is unfortunate that a hearing test was not done between the radical mastoid operation, and the intracranial operation, as this might have helped in assessing the particular effects of the two operations upon the ultimate auditory and vestibular conditions.

CASE NO. 33 — Mr. W. P., age 40. Admitted October 24, 1932.

DIAGNOSIS: Meningeal fibroblastoma of the left temporal fossa.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

October 24, 1932

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Slight loss of hearing to watch test, left ear, to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

NOTE: Diminishing hearing acuity had been noted in the left ear during the previous ten years.

OPERATIVE PROCEDURES:

October 25, 1932 — Ventriculography.

October 26, 1932 — Operator, W. V. Cone. Left temporo-parietal osteoplastic craniotomy with removal of tumor. The tumor filled the entire left middle fossa, extending from the lesser wing of the sphenoid to the petrous pyramid, and medially to the circle of Willis. It measured 7x5x5 cm. and weighed about 90 grams.

April 23, 1936 — The patient is feeling well except for post-operative epilepsy, which began 2½ years after the operation.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 23, 1936

NOSE and THROAT — Chronic infection, tonsillar remnants.

OTOSCOPY — Drums normal.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 9).

Subclass, Right A_3 with dip 4096 d.v. to 35 s.u. Left A_2 with dip 4096 d.v. to 50 s.u.

NOTE: Masking one ear caused very little change in the other.

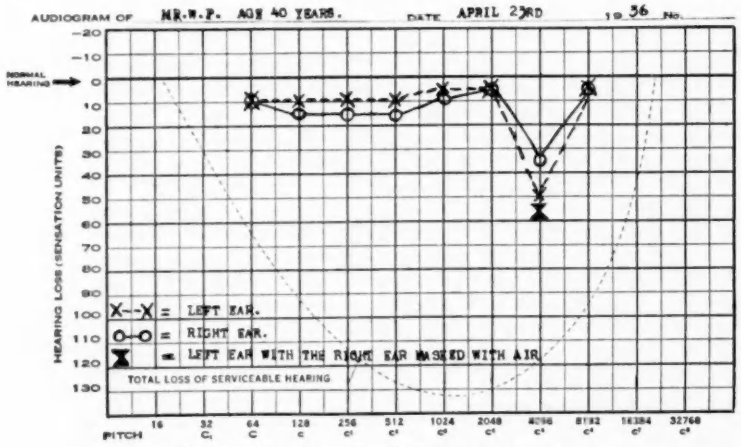


FIG. 9 — (McNally) — Audiogram from case 33.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	25 seconds	25 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for BC or ABC.

Monochord	AC	15,000 d.v.	14,000 d.v.
	BC	16,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: Except for a bilateral dip in the region of 4096 d.v. shown in the audiogram, the post-operative hearing test was essentially normal. The significance of this is not as yet known. This will be discussed with other similar audiograms.

CASE No. 34 — Mr. T. S., age 54. Admitted March 31, 1934.

DIAGNOSIS: Meningeal fibroblastoma, right fronto-parieto-temporal region. Jacksonian Epilepsy.

NOTE: Jacksonian seizures began in 1928, and recurred two months post-operatively. *Aura* — A sense of nausea concomitant with the ringing of bells in both ears.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 31, 1934

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

April 3, 1934 — Encephalography.

April 4, 1934 — Operator, W. V. Cone. Right fronto-parietal osteoplastic craniotomy with complete removal of the tumor. The tumor weighed 90 grams. The dural defect measured 8x8 cm., and was closed with fascia lata. The tumor was located just in front of the pre-central convolution, right, the bulk of it being below the level of the anterior horn and body of the ventricle.

April 30, 1936 — Although he is still troubled with epileptic seizures, the general health of the patient is good.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 30, 1936

NOSE and THROAT — Normal.

OTOSCOPY — *Right Drum*, normal. *Left Drum*, slightly dull and retracted.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3; Left A_3 C_4 to 40 s.u. somewhat similar to Fig. 18.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	70 s.u.	65 s.u.
	ABC	45 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	20 seconds	20 seconds
	ABC	35 seconds	35 seconds

Weber — With BC referred to the right of the midline. Blocking either ear causes the sound to be referred to that side from the vault or forehead.

Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The intracranial lesion has not produced any significant hearing loss, except for the slight high tone loss on the left side which may be partly explained by the patient's age (Fig. 2).

CASE NO. 35 — Mrs. E. S., age 53. Admitted February 25, 1936.

DIAGNOSIS: Glioblastoma multiforme, right occipital region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 25, 1936

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

February 27, 1936 — Operator, W. V. Cone. Bilateral occipital trepanation.

February 29, 1936 — Operator, W. V. Cone. Biopsy.

March 1, 1936 — Operator, W. V. Cone. Right parieto-occipital craniotomy with removal of the tumor. An oval mass of tissue which measured 4.1x3.5x3.5 cm. was removed from the right cerebral hemisphere where the occipital, temporal, and parietal lobes join.

April 23, 1936 — General condition of the patient is good.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 23, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right $A_2 C_4$; Left $A_3 C_4$ (Audiogram very similar to Fig. 18).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	85 s.u.
	ABC	55 s.u.	65 s.u.
c^1 (256)	BC	75 s.u.	75 s.u.
	ABC	55 s.u.	60 s.u.

Tuning Fork Tests			
c^2 (512)	AC	65 seconds	60 seconds
	BC	17 seconds	17 seconds
	ABC	32 seconds	20 seconds

Weber — Referred to the right ear except from the occiput for BC, likewise referred to the right for ABC, but this is not constant, and one cannot be sure of the patient as a witness.

Monochord	AC	14,000 d.v.	10,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The removal of this tumor from the posterior part of the right cerebral hemisphere ordinarily thought to be the area of cortical representation of the cochlea has not had any significant effect upon the patient's hearing. Inasmuch as it is a unilateral lesion this is not surprising. Slight high tone loss on the audiometer and confirmed by the monochord is in keeping with the patient's age (see Fig. 2).

SUMMARY OF GROUP THREE

In this group of nine patients the tumor was situated in the following regions:

Frontal Lobe
- Right ..... Cases 28, 32, 34
- Left ..... Case 27

Frontal Lobes — Bilateral ..... Case 29

Parietal Lobe — Right ..... Case 31

Occipital Lobe
- Right ..... Case 35
- Left ..... Case 30

Temporal — Left ..... Case 33

In each of the seven patients showing some loss of hearing, the patient's age, or evidence of middle ear disease, might be advanced as an alternative explanation to the brain lesion as a cause for the hearing loss, this in spite of the fact that some of these tumors were very large. Several weighed over a hundred grams, and one (case 30) weighed over two hundred grams. Even in this last patient the hearing was practically normal if the patient's age is considered. The tumor was bilateral in only one patient (case 29) and it was located in the frontal areas.

Detailed discussion of the seven patients showing a hearing loss will be found under the following headings:

Cases 30, 31, and 32 — In the impression of the individual case reports

Cases 34 and 35 — Gradual High Tone Loss

Cases 29 and 33 — Audiogram "dip" at 4096 d.v.

GROUP FOUR

Cerebellar Tumors and Abscesses

Each of the six patients in this group had a lesion in the posterior fossa of the brain and three of them showed some loss of hearing. A pre-operative hearing examination in three of the patients did not reveal any hearing loss.

CASE REPORTS

CASE No. 36 — Mr. A. B., age 38. Admitted June 8, 1935.

DIAGNOSIS: Hemangioblastoma, right lateral lobe of the cerebellum.
NOTE: During the attacks of faintness, there was a humming noise in both ears.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

June 8, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Drums normal.

HEARING EXAMINATION

AUDIOGRAM (Similar to Fig. 9) — Subclass A_2 with dip at 4096 to 55 s.u. right and to 40 s.u. left.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c_2 (512)	ABC	60 s.u.	65 s.u.
Tuning Fork Tests c^2 (512)			
Rinné		AC greater than BC	AC greater than BC
Schwabach		+ 10 seconds	+ 10 seconds
Weber — Not lateralized.			
Whispered Voice		10 feet	12 feet

Monochord — Not done.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURES:

June 14, 1935 — Bilateral occipital trepanation.

June 15, 1935 — Ventriculography.

June 15, 1935 — Operator, A. R. Elvidge. Sub-occipital myoplastic craniotomy and removal of cystic cerebellar tumor, about the size of a small orange from the right lateral lobe. It was separated from the ventricle by a very thin layer of tissue.

March, 1936 — Patient has returned to his regular work. Condition improved.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 2, 1936

NOSE and THROAT — Acute nasopharyngitis. Chronic tonsillitis.

OTOSCOPY — Drums are normal.

HEARING EXAMINATION

AUDIOGRAM (Similar to Fig. 9) — Subclass A_2 with 4096 dip right and left down to 45 s.u. right and to 35 s.u. left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	60 s.u.	65 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	55 s.u.
Tuning Fork Tests			
c^2 (512)	AC	65 seconds	65 seconds
	BC	20 seconds	22 seconds
	ABC	27 seconds	27 seconds
Weber — Not done.			
Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	18,000 d.v.	18,000 d.v.
	ABC	18,000 d.v.	19,000 d.v.
Watch Test		12/15″	12/15″
Whispered Voice		8 feet	8 feet

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The removal of the cerebellar tumor has not caused any appreciable change in the hearing. The significance of the dip at 4096 d.v. will be discussed with the other cases showing a similar audiogram.

CASE No. 37 — Mr. E. H., age 37. Last admission May 1, 1936.

DIAGNOSIS: Cerebellar tumor (astrocytoma), left lateral lobe.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

August 15, 1932

NOSE and THROAT — Not examined.

OTOSCOPY — Not done.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

August 16, 1932 — Operator, W. V. Cone. Suboccipital craniotomy and complete removal of tumor and cyst wall from the left cerebellar lobe. The cyst contained 40 cc. of fluid.

May 1, 1936 — Patient re-admitted for symptoms suggestive of recurrence of the tumor.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 4, 1936

NOSE and THROAT — Normal except for a slight mucosal congestion of the nose.

OTOSCOPY — Drums normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2 C_6 down to 60 s.u.; Left A_2 C_6 down to 60 s.u. (Similar to left ear in Fig. 10).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	65 s.u.	60 s.u.
	ABC	55 s.u.	50 s.u.
c^1 (256)	BC	60 s.u.	60 s.u.
	ABC	45 s.u.	55 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	25 seconds	25 seconds
	ABC	40 seconds	40 seconds
Weber — Not referred for BC or ABC.			
Monochord	AC	15,000 d.v.	15,000 d.v.
	BC	15,000 d.v.	15,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The high tone loss in the absence of any local, middle or internal ear disease may possibly be due to the intracranial lesion. Its significance will be discussed in conjunction with other cases showing the same type of audiogram.

CASE NO. 38 — Master H. P., age 5½. Admitted March 5, 1935.

DIAGNOSIS: Medulloblastoma, left cerebellar hemisphere.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 5, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — Spontaneous vertical nystagmus upwards.

Cold Caloric Test — Perversion of the nystagmus from the vertical canals, while the response from the horizontal canals shows some diminution of the quick phase of the nystagmus.

OPERATIVE PROCEDURES:

March 6, 1935 — Operator, W. V. Cone. suboccipital craniotomy with removal of the tumor, which occupied almost the entire left lateral lobe of the cerebellum.

April 13, 1936 — Great general improvement. Walks very well.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 13, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, right and left.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	60 s.u.	55 s.u.

c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests

c^2 (512)	AC	65 seconds	65 seconds
	BC	25 seconds	25 seconds
	ABC	35 seconds	35 seconds

Weber — Not referred for either BC or ABC.

Monochord	AC	17,000 d.v.	17,000 d.v.
	BC	18,000 d.v.	18,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The removal of the cerebellar tumor has resulted in no significant hearing loss. Inasmuch as pre-operative hearing tests were not done accurately, estimation of improvement of hearing post-operatively cannot be determined. It is interesting to note that all of the abnormal vestibular signs have cleared up.

CASE No. 39 — Mr. A. R., age 18. Admitted May 31, 1935.

DIAGNOSIS: Cystic astrocytoma, cerebellum, mid-line.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

June 12, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right B_1; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	ABC	55 s.u.	60 s.u.

Tuning Fork Tests — Not done.

Monochord — Not done.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURES:

June 14, 1935 — Operator, W. V. Cone. Bilateral occipital trepanation.

June 19, 1935 — Operator, W. G. Penfield. Suboccipital craniotomy and removal of tumor. The specimen measured 5.5 cm. in diameter and was roughly spherical. Total weight of solid parts was 48 grams. It completely filled the fourth ventricle, and had displaced and destroyed at least half of the cerebellum.

March 2, 1936 — Condition greatly improved, walks without staggering. Feels well.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 2, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	75 s.u.
	ABC	65 s.u.	60 s.u.
c^1 (256)	BC	65 s.u.	65 s.u.
	ABC	60 s.u.	60 s.u.
Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	30 seconds	25 seconds
	ABC	37 seconds	30+ seconds
Weber — Not recorded.			
Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	18,000 d.v.	18,000 d.v.
	ABC	19,000 d.v.	19,000 d.v.
Watch Test		15/15″	15/15″
Whispered Voice		8 feet	8 feet

VESTIBULAR EXAMINATION — Very marked irregular spontaneous nystagmus.
Cold Caloric Test — Normal.

IMPRESSION: It is interesting to note that in spite of the large amount of brain destruction in the posterior fossa, the hearing is very litttle affected.

CASE No. 40 — Mr. F. S., age 16.. Admitted February 9, 1935.

DIAGNOSIS: Abscess, left cerebellar hemisphere, streptococcus hemolyticus, following acute streptococcus hemolyticus mastoiditis.

NOTE: The patient had an upper respiratory infection December 30, 1935, and a discharging left ear January 5, 1936. Simple mastoidectomy left was performed January 25, 1936, and repeated a week later, but symptoms of vomiting and ataxia continued, and a few days prior to his admission to the Montreal Neurological Institute, he was troubled with dimness of vision.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 9, 1935

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Normal.
Left Drum — Left canal filled with purulent discharge. Granulating wound left mastoid region.

HEARING EXAMINATION — Left sided impairment of hearing to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

1. February 9, 1935 — Operator, W. V. Cone. Bilateral occipital trepanation and ventricular puncture.
2. February 10, 1935 — Operator, W. V. Cone. Suboccipital craniotomy and drainage of abscess of left cerebellar hemisphere. The abscess was 2 cm. below the surface and contained about 1½ ounces of pus. April 23, 1936 — The patient has made a complete recovery.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 23, 1936

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Normal.
Left Drum — Slight thickening. Well-healed left mastoid scar.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	60 s.u.	60 s.u.
	ABC	50 s.u.	50 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	45 s.u.	45 s.u.

Tuning Fork Tests

c^2 (512)	AC	65 seconds	65 seconds
	BC	35 seconds	35 seconds
	ABC	40 seconds	40 seconds

Weber — Not referred for BC or ABC.

Monochord			
	AC	15,000 d.v.	14,000 d.v.
	BC	17,000 d.v.	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: The acute middle ear infection, followed by a simple mastoid operation and damage to the cerebellar tissue has not caused any significant loss of hearing.

CASE NO. 41 — Miss J. S., age 32. Admitted March 22, 1935.

DIAGNOSIS: Meningeal fibroblastoma, left cerebellar pontine angle tumor.

NOTE: The patient has had indefinite ear trouble in childhood with otorrhea in one ear seven years ago. Headache, rotatory vertigo, nausea and vomiting present for five years and ringing in the left ear for one month.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 26, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Right drum shows a chalk deposit along the antero-interior edge. Both drums are retracted.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left B_1.

(Unfortunately during this test, the two notes, 4096 d.v. and 8192 d.v., went out of order on the audiometer and could not be tested.)

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC			
c^2 (512)	BC	60 s.u.	65 s.u.
Tuning Fork Tests c^2 (512)			
Rinné		AC greater than BC	AC greater than BC
Schwabach		Normal	Normal
Weber — Not lateralized for BC or ABC.			
Monochord	AC	15,000 d.v.	14,000 d.v.
Watch	AC	20/25″	0/25″
Whisper		8 feet	At ear
(Op. ear masked with B.M.)			

VESTIBULAR EXAMINATION — A slight spontaneous horizontal rotatory nystagmus in the end-points.

Cold Caloric Tests — Normal.

Rotation Tests — Normal.

NOTE: Above tests not done during present investigation.

OPERATIVE PROCEDURES:

March 29, 1935 — Ventriculography.

March 29, 1935 — Operator, W. G. Penfield. Suboccipital craniotomy with removal of tumor. The tumor weighed 5.3 grams. It was a cauliflower mass measuring 4.5x3.5x1 cm. and was attached to the undersurface of the tentorium and to the dura of the floor of the posterior fossa. It surrounded the seventh and eighth nerves and lay largely in the internal auditory meatus. It was also partly attached to the fifth nerve. The tumor was removed without obvious damage to the 5th, 7th and 8th nerves.

May 13, 1936 — Satisfactory general condition, no facial paralysis, but some slight ataxia.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 3, 1935

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right B_1; Left E_1 C_6 down to 75 s.u.

(This entire audiogram is very similar to that shown in Fig. 10, done one year later.)

It is most unfortunate that the two notes 4096 d.v. and 8192 d.v. had not been tested pre-operatively, because it is now impossible to attach any significance to the dip shown in the left ear.

BONE CONDUCTION TESTS

	RIGHT	*LEFT*
Tuning Fork Tests c^2 (512)		
Rinné	AC greater than BC	AC greater than BC
Whisper	25 feet	20 feet
Conversational Voice	40 feet	30 feet

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

NOTE: The above tests not done by the present investigators.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 13, 1936

NOSE and THROAT — Normal.

OTOSCOPY — As before operation.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 10).

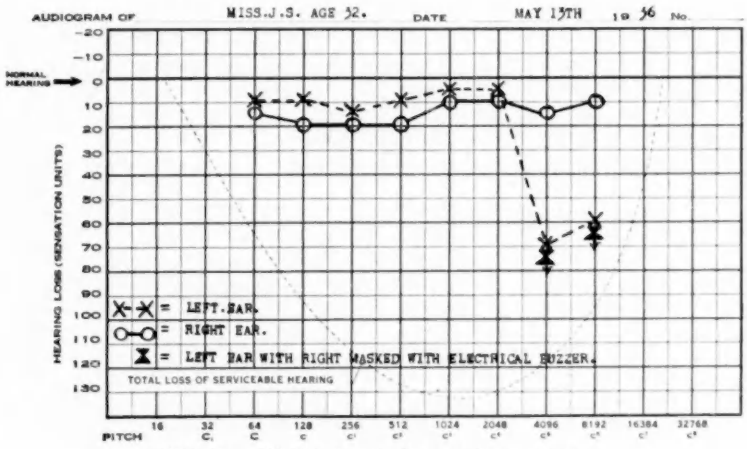


FIG. 10 — (McNally) — Audiogram from case 41.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with E.M.	No masking	Op. ear masked with E.M.
c^2 (512)	BC	70 s.u.		70 s.u.	
	ABC	50 s.u.		50 s.u.	
c^1 (256)	BC	65 s.u.		65 s.u.	
	ABC	45 s.u.		45 s.u.	
Tuning Fork Tests					
c^2 (512)	AC	75 seconds		75 seconds	
	BC	15 seconds	3 seconds	30 seconds	11 seconds
	ABC	50 seconds		55 seconds	
Weber — Not referred to either ear from any place on the head.					
Monochord	AC	13,000 d.v.	15,000 d.v.	14,000 d.v.	12,000 d.v.
	BC	14,000 d.v.	17,000 d.v.	15,000 d.v.	15,000 d.v.

NOTE: It is interesting to note that for ABC in the right ear, that blocking only the right ear results in an ABC of 35 seconds from the mastoid, whereas blocking only the the left ear, the fork on the right mastoid was heard for 50 seconds. It is difficult to attach any significance to these findings other than that bone conduction is better in the left ear. Masking, which we have found to be ineffective for the monochord has had the peculiar effect of raising the hearing in the right ear for AC and BC by 2,000 d.v. and lowering it in the left ear for AC by the same amount. It did not affect the BC in the left ear. This has been noted in a few other cases.

VESTIBULAR EXAMINATION — There was a spontaneous nystagmus to the right, horizontal rotatory, small amplitude and fairly well sustained.

Cold Caloric Test — Elicited a normal response from the vertical and horizontal canals of each ear, but the response from the right ear was slightly exaggerated. This was slight but, nevertheless, quite definite.

IMPRESSION: In spite of the gross lesion in the region of the left eighth nerve, the hearing has definitely improved post-operatively for the low notes from 64 d.v. to 2048 d.v., inclusive; the hearing now being better

in the left ear than in the right for this range. Because the two highest notes were not tested pre-operatively, it is impossible to assess the significance of the dip in the left high notes. If present pre-operatively, it may have been due to the old middle ear lesion, or it may have been due to the presence of the tumor. The significance of the alteration for fork c^2 (512) in the right ear is equally obscure. The slight exaggeration of response from the right labyrinth is probably associated with the intracranial lesion.

SUMMARY OF GROUP FOUR

In this group of six patients the lesion was situated in the following regions:

Cerebellar Hemisphere
- Right ..... Case 36
- Left ..... Cases 37, 38, 40

Cerebellum — Mid-line ..... Case 39

Cerebello-Pontine Angle — Left ..... Case 41

The hearing loss shown by any patient in this group was very slight except in the central case in which the lesion was in the cerebello-pontine angle. It is particularly interesting to note that in this patient the hearing improved after the tumor was removed. The vestibular tests were carried out pre-operatively in four of the patients but only in case 38, a large tumor of the left hemisphere, was there any abnormality shown. The reactions returned to normal after removal of the tumor.

Detailed discussion of the three patients showing a hearing loss will be found under the following headings:

Case 36 — Audiogram "Dip" at 4096 d.v.

Cases 37, 41 — Abrupt High Tone Loss

* * *

The last three groups are made up of patients who have had either brain tissue removed to eliminate cerebral cicatrices, cerebral tumors removed or else they have had cerebellar tumors removed. Though there have been slight hearing losses shown by the tests in some of the patients, in no patient except case 41 was the hearing loss sufficient to be classed as deafness unless there was very definite evidence of gross middle ear disease. In no other patient could the hearing loss be considered diagnostic of the brain lesion (see subsequent remarks about special audiograms).

GROUP FIVE

Perineural Fibroblastomas of the Eighth Nerve

There were four cases of eighth nerve tumor in this group. The pre-operative otolaryngological examination was done in each patient but not during the present investigation. In only one of the patients did we have the opportunity of carrying out a post-operative hearing examination.

CASE REPORTS

CASE NO. 42 — Miss E. G., age 20. Admitted October 24, 1934.

DIAGNOSIS: Perineural fibroblastoma left eighth nerve.

NOTE: Tinnitus, deafness and dizziness in the left ear for seven months.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

October 25, 1934

NOSE and THROAT — Tonsils large, buried and filled with débris.

OTOSCOPY — No history of ear discharge. Drum membranes are dull, atrophic and somewhat retracted.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_3; Left F_3.

BONE CONDUCTION TESTS

Tuning Fork Tests c^2 (512)	*RIGHT*	*LEFT*
Weber — Referred to the right.		
Rinné	AC = BC	AC greater than BC
Schwabach	Normal	— 10 seconds
Whispered Voice	20 feet	1 foot
Conversational Voice	35 feet	5 feet

Monochord — Not tested.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

NOTE: The above examinations were not done by the present investigators.

OPERATIVE PROCEDURES:

October 27, 1934 — Operator, W. V. Cone. Suboccipital craniotomy with complete removal of left eighth nerve tumor. The seventh and eighth nerves were completely evulsed. A portion of the left lateral lobe of the cerebellum was removed. The tumor had so enlarged the porous acousticus that the tip of the index finger could be placed in it.

November 7, 1934 — Anastomosis of the facial and hypoglossal nerves of the left side.

November 16, 1936 — Patient's general condition is good.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 16, 1936

NOSE and THROAT — Some evidence of chronic septic tonsillitis.

OTOSCOPY — A suggestion of a small scar immediately behind the right malleus. Slight retraction of both drums, otherwise normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left B_3.

(Masking right ear with electrical buzzer full strength eliminates hearing in left ear for all audiometer notes.)

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with E.M.	No masking	Op. ear masked with E.M.
c^2 (512)	BC	75 s.u.	85 s.u.	60 s.u.	50 s.u.
	ABC	60 s.u.		60 s.u.	
c^1 (256)	BC	60 s.u.	75 s.u.	60 s.u.	60 s.u.
	ABC	55 s.u.		55 s.u.	

	RIGHT			*LEFT*		
Tuning Fork Tests c^2 (512)	No masking	Quiet room	Op. ear masked with E.M.	No masking	Quiet room	Op. ear masked with E.M.
AC	70 seconds	65 seconds	40 seconds	3 seconds	12 seconds	5 seconds
BC	15 seconds	15 seconds	10 seconds	22 seconds	22 seconds	55 seconds
ABC	20 seconds			32 seconds		

Weber — With either BC or ABC the sound is as likely to be referred to the left as to the right ear.

Monochord	*RIGHT*			*LEFT*		
AC	17,000 d.v.	18,000 d.v.	18,000 d.v.	16,000 d.v.	17,000 d.v.	16,000 d.v.
BC	19,000 d.v.	18,000 d.v.	22,000 d.v.	18,000 d.v.	18,000 d.v.	20,000 d.v.

NOTE: It is interesting to note that for audiometer BC the tuning fork tests and the monochord, masking the normal ear has increased the hearing on the side of the avulsed eighth nerve.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — There was a slight spontaneous nystagmus at the end point on gaze to either side, which suggested a fixation nystagmus. The right labyrinth reacted normally to the cold caloric test. The left ear showed no response to 10 cc. of ice water slowly introduced.

IMPRESSION: If this patient were being examined by an otologist and her previous history was not known, the audiogram would suggest a high grade nerve deafness in the left side. The effect of masking the right ear would be to suggest complete nerve deafness on the left side. This diagnosis would be confirmed by the caloric tests. On the other hand, the bone conduction tests, if carried out by the audiometer would suggest normal bone conduction in each ear with possibly a slight conduction lesion on the left side. The effect of masking on this test would suggest some nerve deafness on the right side. The further checking of the bone conduction with the fork tests suggests some middle ear deafness on the left side with a slight falling off of bone conducton on the right side and early nerve deafness right. The effect of masking for this particular test would heighten the suspicion of nerve deafness on the right side. The monochord test suggests a normal range of hearing for both air conduction and bone conduction and in both ears. The Weber suggests that air conduction and bone conduction are equal on both sides. To summarize, the audiogram and the vestibular tests suggest complete destruction of the left eighth nerve. The bone conduction tests, whether by audiometer or by fork suggest a very mild bilateral deafness of middle ear character on the left, and an early nerve deafness on the right. The Weber suggests symmetrical hearing on both sides, and the monochord suggests that it is within normal limits.

NOTE: This patient was seen by all three examiners, who agreed that her mental condition was clear and that she was a satisfactory witness. Since the seventh and eighth nerves were completely evulsed it is hard to imagine that there has been any regrowth of the eighth nerve. It is the bone conduction tests and monochord which are most misleading and both are difficult to exclude from the non-tested ear.

CASE NO. 43 — Mr. M. H., age 26. Admitted May 11, 1934.

DIAGNOSIS: Perineurial fibroblastoma, right eighth nerve.

NOTE: Bilateral otitis media in infancy. Deafness in the right ear for 9 years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 12, 1934

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Slight retraction.
Left Drum — Scarring in the region of the short process of the malleus.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 11).

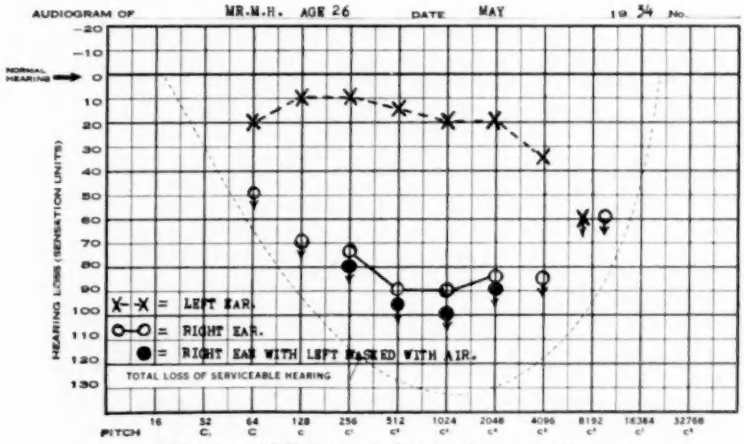


FIG. 11 — (McNally) — Audiogram from case 43.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC c² (512)	BC	90 s.u.	65 s.u.

NOTE: BC in right ear still heard when left ear is masked with B.M.

Tuning Fork Tests c² (512)

	BC greater than AC	AC greater than BC
Rinné		
Schwabach	+15 second	—20 seconds

Weber — Not lateralized.

NOTE: Bone conduction is apparently heard over the right mastoid, but blocking the right canal does not increase the loudness, whereas blocking the left canal does, suggesting that the sound is actually being heard by BC across in the left ear.

		RIGHT	*LEFT*
Watch Test		0/20″	15/20″
Whispered Voice		N.H.	15 feet
Loud Conversation		1 foot (Left ear not masked)	
Monochord	AC	N.H.	15,000 d.v.

VESTIBULAR EXAMINATION — Marked spontaneous nystagmus in all eye positions.

Cold Caloric Test — Right, No reaction; Left, normal.
Rotation Test — Symmetrical reaction right and left.

NOTE: Above tests done before present investigation was undertaken.

OPERATIVE PROCEDURE:

May 12, 1934 — Operator, Wilder Penfield. Suboccipital craniotomy and complete removal of tumor of the right eighth nerve.

August 29, 1935 — Patient is in good general condition.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

Not obtained.

IMPRESSION: The amount of deafness shown to be present in the left ear could be explained by the ear trouble, which occurred in early childhood. The small amount of hearing in the right ear which could still be detected by the audiometer when the left ear was masked may possibly have been due to the fact that the right eighth nerve had not been completely destroyed by the tumor. This suggestion is further borne out by the fact that there was apparently some bone conduction from the right ear, when the left was masked out by a flow of air. The fact, however, that the fork on the right mastoid was heard for the most part by the left ear was demonstrated when occluding the left ear canal increased the hearing for the fork on the right mastoid. It is very difficult to understand why the monochord was not heard by air conduction in the diseased ear in this patient and in case 44, when it was heard so readily in cases 42 and 48, in each ear of whom one eighth nerve had been sectioned at operation.

CASE NO. 44 — Mrs. M. L., age 30. Admitted November 14, 1935.

DIAGNOSIS: Perineurial fibroblastoma, right auditory nerve.

NOTE: Tinnitus and increasing deafness right ear for 3 years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

November 19, 1935

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 12)

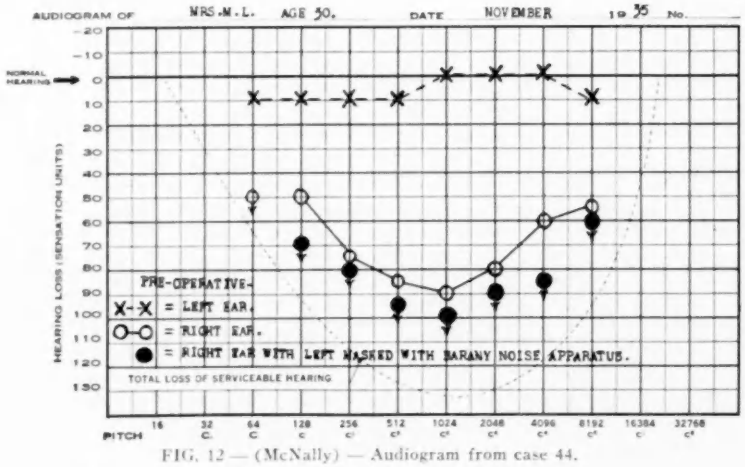


FIG. 12 — (McNally) — Audiogram from case 44.

BONE CONDUCTION TESTS

		RIGHT	*LEFT*
Audiometer BC		Not done	Not done
c^2 (512)			
Tuning Fork Tests			
Weber — Referred to the left.			
c^2 (512)	ABC	0	Normal
(Fork over right mastoid heard in left ear.)			
Monochord	AC	0	16,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Right, no response. Left, normal.

NOTE: The above tests were done before the present investigation was undertaken.

OPERATIVE PROCEDURES:

November 21, 1935 — Operator, Dr. W. V. Cone. Bilateral occipital trepanation and ventriculography.

November 23, 1935 — Operator, Dr. W. V. Cone. Right myoplastic suboccipital craniotomy with complete removal of tumor of the right eighth nerve. The tumor was situated in the internal auditory meatus and had enlarged it considerably. The tumor weighed 28 grams. The seventh and eighth nerves were removed with the tumor.

Note: The patient died the day following the operation.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

Not obtained.

IMPRESSION: A very difficult finding to understand in view of case 42 and case 48 is that in this patient the monochord was heard up to 16,000 d.v. in the left ear, and not heard at all in the right or diseased ear. The vestibular tests, the audiogram, the fork tests, including the Weber and the monochord, were all in keeping with destruction of the right eighth nerve.

CASE No. 45 — Miss E. M., age 47. Admitted December 17, 1929.

DIAGNOSIS: Left cerebello-pontine angle tumor (perineurial fibroblastoma) involving the eighth nerve.

NOTE: Deafness and tinnitus left ear over a period of four years.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

September 20, 1929

NOSE and THROAT — Normal.

OTOSCOPY — Normal except for a slight retraction of the drums.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 13).

BONE CONDUCTION TESTS

	RIGHT	*LEFT*
Audiometer BC	Not done	Not done
Tuning Fork Tests c^2 (512)		
Schwabach	Normal	—15 seconds
Weber — Referred to the right.		
Rinné	AC greater than BC	AC = BC (Both reduced)
Whispered Voice	20 feet	Not heard

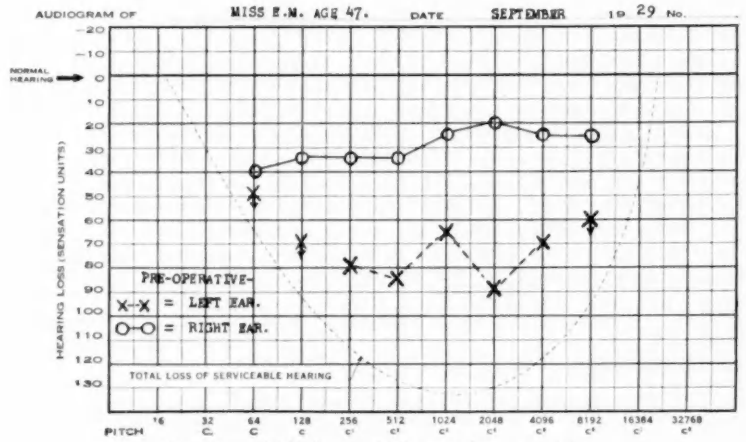


FIG. 13 — (McNally) — Audiogram from case 45.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Right, Perversion of response from the vertical canals. Left, No reaction.

NOTE: Tests done before the present investigation was undertaken.

OPERATIVE PROCEDURE:

September 23, 1929 — Operator, Dr. Wilder Penfield. Suboccipital craniotomy and removal of tumor from the left eighth nerve. The neoplasm was about the size of a plum.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

Not obtained.

IMPRESSION: It is quite possible that had the right ear been masked, the left ear would have been shown to be completely deaf. This is one of the few cases where the Weber was referred to the ear which showed the best bone conduction.

SUMMARY OF GROUP FIVE

Each of the four patients in this group had a tumor of the eighth nerve and in every instance the pre-operative tests showed a high grade nerve deafness on the side of the tumor. The tests were accompanied by masking in only two of the patients, cases 43 and 44. Any hearing shown on the audiogram for the diseased ear was completely eliminated by masking the normal ear. In two patients the Weber was referred to the diseased side but in two others it was not consistently referred to either side in spite of extensive nerve damage shown at operation. The monochord was used in two patients only, cases 43 and 44, and then only for air conduction. It was not heard at all in the diseased ear but was

heard within normal limits in the healthy ear. This is surprising in view of the loudness of the note from the monochord which ordinarily may be heard in the opposite ear. Furthermore, in case 42, which was examined post-operatively, the monochord was heard within normal limits in the ear from which the eighth nerve had been evulsed; in this patient, masking the normal ear with an electric buzzer actually increased the hearing for the monochord in the diseased ear (see also case 48, group six).

During the pre-operative examination the vestibular reactions were found to be absent on the diseased side in every patient and in case 45 the reactions of the opposite side were perverted.

GROUP SIX

Miscellaneous Cases

This group consists of five patients that do not properly belong to any of the other groups. It includes a tumor of the basal ganglia, a tumor of the third ventricle, a case of bilateral sub-dural effusion in a patient with bilateral chronic suppurative otitis media, a case of section of one eighth nerve for Ménière's Syndrome, and lastly a case of osteoplastic craniotomy included as a partial control of group one.

CASE REPORTS

CASE NO. 46 — Mr. H. F., age 23. Admitted September 27, 1935.

DIAGNOSIS: Astrocytoma basal ganglia, left.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

September 27, 1935

NOSE and THROAT — Normal to superficial examination.

OTOSCOPY — Normal.

HEARING EXAMINATION — Normal to superficial examination.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.

OPERATIVE PROCEDURES:

October 3, 1935 — Encephalography.

October 10, 1935 — Operator, W. Penfield. Left osteoplastic craniotomy and removal of tumor. The neoplasm was found lying in the left caudate nucleus and thalamus, about the size of a large plum.

March 10, 1936 — Patient had his first epileptic seizure. His general condition is quite satisfactory.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

February 24, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass, Right A_2; Left A_2.

BONE CONDUCTION TESTS

Audiometer BC		RIGHT	*LEFT*
c^2 (512)	BC	70 s.u.	70 s.u.
	ABC	45 s.u.	55 s.u.
c^1 (256)	BC	70 s.u.	70 s.u.
	ABC	50 s.u.	50 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	25 seconds	25 seconds
	ABC	35 seconds	30 seconds

Weber — Not lateralized for BC or ABC.

Watch Test		6/6″	6/6″
Whispered Voice		10 feet	10 feet
Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	17,000 d.v.
	ABC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Not done.
Rotation Test — Normal.

IMPRESSION: The removal of the tumor from the basal ganglia has not adversely influenced the hearing.

CASE NO. 47 — Mr. L. G., age 39. Admitted February 7, 1936.

DIAGNOSIS: Epilepsy. Chronic subdural effusion, bilateral. Chronic suppurative otitis media, bilateral. Right quiescent. Left active with cholesteatoma.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 20, 1936

NOSE and THROAT — Normal.

OTOSCOPY

Right — There is a large kidney-shaped perforation involving the lower two-thirds of the drum; the upper portion of the drum, including the long handle of the malleus, is intact. There is no evidence of moisture. The niche in the round window can be clearly seen.

Left — The lower part of the drum shows a healed perforation. There is a large fistula leading from the roof of the canal into the attic region filled with foul-smelling discharge and débris suggesting cholesteatoma. The fistula involves practically the whole of the outer attic wall.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 14).

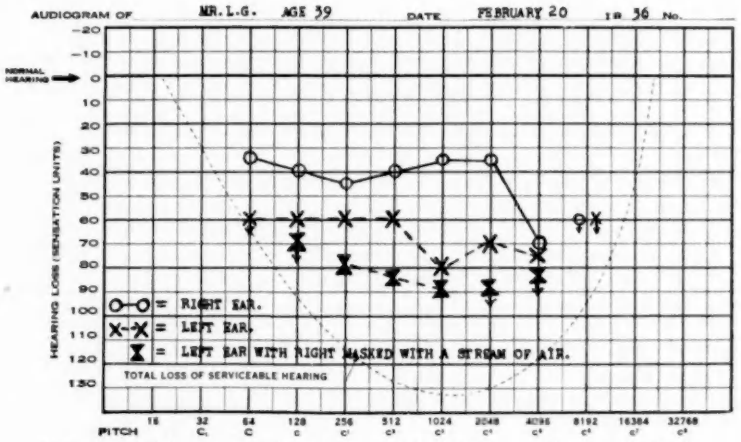


FIG. 14 — (McNally) — Audiogram from case 47 (Pre-operative examination).

BONE CONDUCTION TESTS

		RIGHT	LEFT
Audiometer BC			
c^2 (512)	BC	60 s.u.	50 s.u.
	ABC	55 s.u.	45 s.u.
c^1 (256)	BC	55 s.u.	45 s.u.
	ABC	45 s.u.	35 s.u.
Tuning Fork Tests			
c^2 (512)	AC	20 seconds	
	BC	35 seconds	55 seconds
	ABC	40 seconds	55 seconds
Weber — Not referred for BC or ABC.			
Monochord	AC	9,000 d.v.	6,000 d.v.
	BC	16,000 d.v.	13,000 d.v.
Watch Test		0/20″	0/20″
Ordinary Conversation		3 feet	Not heard

VESTIBULAR EXAMINATION

Cold Caloric Test — Right Ear: Cold air douche for 45 seconds with head back (horizontal canal) produced a nystagmus to the left, but this was diagonally upwards and to the left in both eyes, in the plane of the right anterior vertical canal. When the head was moved forward to the ordinary head erect position (vertical canals), there was a normal rotatory nystagmus to the left. No past pointing.

Left Ear: No response obtained to 15 cc. of ice water slowly introduced, nor to one minute of cold air douching. No past pointing.

Rotation Test — 10 times to the left produced perfectly normal nystagmic response from the horizontal canal. 10 times to the right produced a similar but minimal response with a few twitches only. The perversions previously noticeable with caloric tests were not confirmed.

NOTE: Two examiners were tested under similar conditions and BC was 25 seconds, ABC 35 seconds. The patient was repeatedly checked and the entire test repeated in a quiet room the following day with very little change in the above findings. BC was

heard in both ears for a longer time than by the examiners and much longer in the left ear, which was shown by the audiometer to have lost all serviceable hearing. Another extraordinary thing is that in spite of marked increase of BC in the left, the Weber was not referred by either BC or ABC. Masking the right ear with a stream of air reduced hearing for the fork on the left mastoid but did not eliminate it. When the fork was on the left mastoid, a masking stream of air introduced into the left ear canal did not appreciably reduce the hearing for this fork. This would suggest that either the fork was heard in the right ear or else that the stream of air was not effective in masking the left ear in the presence of the active middle ear disease on this side. When the fork was on either mastoid, the blocking of either external canal did not appreciably increase the hearing.

OPERATIVE PROCEDURES:

February 26, 1936 — Operator, Dr. W. J. McNally. Radical mastoid operation for removal of cholesteatoma, left middle ear and mastoid.

INTERIM POST-OPERATIVE EXAMINATION

March 3, 1936

HEARING EXAMINATION — Fork c^2 (512) in the right ear as before operation; in the left ear, AC was not heard. To test BC on the left side, a fork which had previously been sterilized, was placed in direct contact with the mastoid bone exposed in the operative wound. In this position, it was heard for 50 seconds. When placed on the edematous tissue one-half inch from the edge of the operative wound, the fork was heard for 45 seconds. This would suggest that the loss in sound conduction due to edematous soft tissue was only five seconds. In direct contact with the bone the fork was heard for five seconds less than when tested pre-operatively over the skin of the intact mastoid. The Weber was not referred in spite of this increased bone conduction in the left ear, and with the left ear heavily swathed in dressings.

OPERATIVE PROCEDURES:

March 15, 1936 — Second stage plastic closure of left radical mastoid operation wound. Dr. W. J. McNally.

April 8, 1936 — Operator, Dr. Wilder Penfield. Bilateral subtemporal decompression and subdural drainage. Considerable amount of subdural fluid removed, more from the right side.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

April 23, 1936

NOSE and THROAT — As before operation.

OTOSCOPY

Right Drum — As before.
Left Drum — Dry and healed radical mastoid cavity. Healed mastoid scar.

BONE CONDUCTION TESTS

Audiometer BC		RIGHT	*LEFT*
c^2 (512)	BC	50 s.u.	55 s.u.
	ABC	50 s.u.	55 s.u.
c^1 (256)	BC	50 s.u.	45 s.u.
	ABC	50 s.u.	45 s.u.
Tuning Fork Tests			
c^2 (512)	AC	12 seconds	
	BC	40 seconds	40 seconds
	ABC	40 seconds	40 seconds

NOTE: Masking right ear with current of air completely eliminates BC for left ear, while masking left has no effect on BC right.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 15).

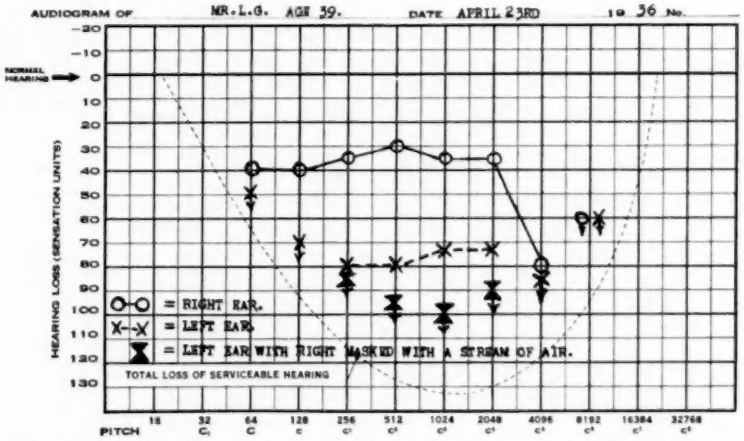

AUDIOGRAM OF MR. L.G. AGE 39. DATE APRIL 23RD 19 36 No.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
= RIGHT EAR.
= LEFT EAR.
= LEFT EAR WITH RIGHT MASKED WITH A STREAM OF AIR.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

FIG. 15 — (McNally) — Audiogram from case 47 (Post-operative examination).

Weber — Weber in the midline is referred to either side, when moved to the right is heard on that side and similarly when moved to the left is heard on the left side. Blocking the right ear alone does not cause the fork to be referred to the right ear from the midline or beyond towards the left side. Blocking the left ear alone does not alter the reference of the Weber. When the fork is in the neighborhood of the left ear, masking the left ear with a current of air may eliminate the sound altogether, but does not cause it to be referred to the opposite side.

		RIGHT	*LEFT*
Monochord	AC	9,000 d.v.	0
	BC	16,000 d.v.	10,000 d.v.

Monochord BC eliminated in left ear by masking with a current of air on right side.

VESTIBULAR EXAMINATION — Same as pre-operatively.

IMPRESSION: This case demonstrates the importance of masking both for the audiometer and for bone conduction tests, whether carried out with the audiometer or by the fork, and for the monochord. In our experience a stream of air, when very strong, has more of a masking effect upon the monochord than has the buzzer type of masker. Before the radical mastoid operation, the audiogram with masking shows almost complete deafness in the left ear, and after the radical mastoid operation there is complete deafness in the left ear. The fork tests c^2 (512) show increased bone conduction which was more marked in the left ear. This marked increase of bone conduction was present even in a quiet room and though apparently coming from the left or more diseased ear, the masking showed that it most likely came from the right or quiescent ear. We have no adequate explanation to offer for this. The hearing was apparently not affected by the removal of the subdural collection of fluid, unless the disappearance of any remnant of hearing from the left ear could be ascribed to the removal of the subdural collection of fluid and not to the radical mastoid operation.

It is interesting to note that bone conduction, whether tested on bare bone or through œdematous soft tissue, varied by only five seconds.

Case No. 48 — Mrs. M. H., age 46. Admitted June 1, 1933.

Diagnosis:

1. Chronic suppurative otitis media, right (healed after radical mastoid operation).
2. Ménière's syndrome.

Note: Intermittent discharge from the right ear since the age of twelve years. Vertigo and tinnitus for 8 years. Radical mastoid operation done on the right ear in 1932. The radical mastoid cavity healed completely, but there was no improvement in the vertigo or head noise.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

February 6, 1933

NOSE and THROAT — Normal.

OTOSCOPY — Healed radical mastoid cavity on the right side. Left ear drum normal.

HEARING EXAMINATION

Tests have been made at repeated intervals, the last having been done on February 6, 1933.

AUDIOGRAM — (See Fig. 16).

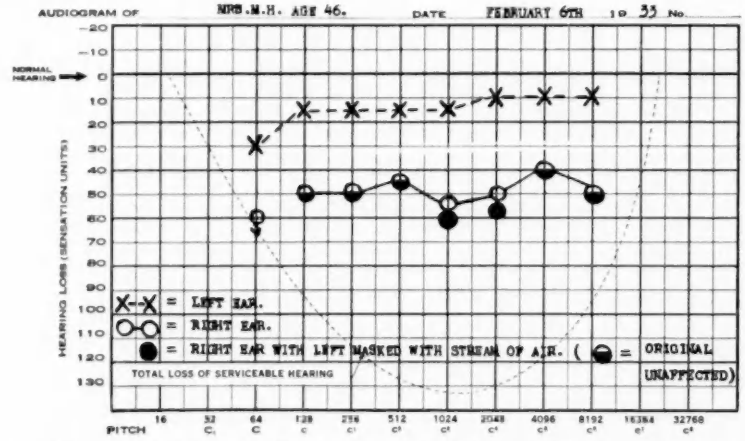


FIG. 16 — (McNally) — Audiogram from case 48 (Pre-operative examination).

BONE CONDUCTION TESTS

		RIGHT	LEFT
Audiometer BC			
c^2 (512)	ABC	55 s.u.	55 s.u.
Tuning Fork Tests c^2 (512)			
Weber — Definitely referred to the right.			
Rinné		BC greater than AC	AC greater than BC
Schwabach		+15 seconds	—15 seconds
Monochord	AC	13,500 d.v.	15,500 d.v.
Watch		Contact	20/30″

VESTIBULAR EXAMINATION

Cold Caloric Test — Left ear normal reaction. Right ear ethyl chloride produces an atypical nystagmoid movement of the eyes, but there was very marked dizziness.

Rotation Tests — Head Erect — Horizontal nystagmus occurred after turning to either side.

Tilting Tests — Normal prompt reaction to slow or quick tilting (patient on hands and knees on the table).

OPERATIVE PROCEDURES:

June 9, 1933 — Operator, W. G. Penfield. Suboccipital craniotomy and right acoustic rhizotomy. Care was taken not to section the right seventh nerve.

May 7, 1936 — The head noises have been considerably less since the section of the right eighth nerve. The dizziness, however, has persisted and the patient has the greatest difficulty in carrying out her routine household duties. Repeated neurological examinations suggest that there is a functional element in the case.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 7, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Healed radical mastoid cavity on the right side. Left ear drum normal.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 17).

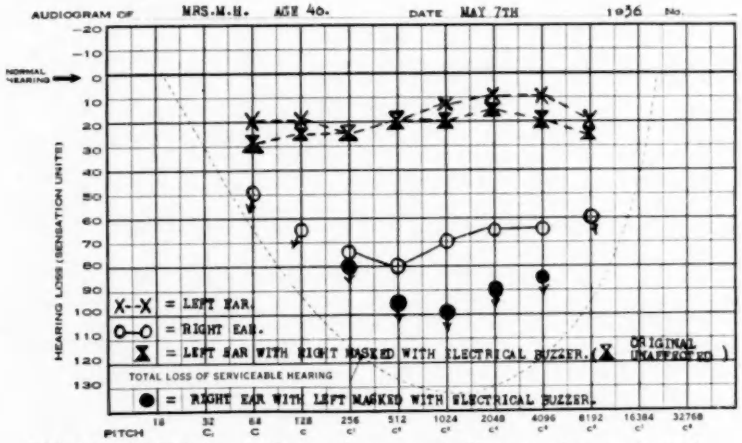


FIG. 17 — (McNally) — Audiogram from case 48 (Post-operative examination).

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*		*LEFT*	
		No masking	Op. ear masked with E.B.	No masking	Op. ear masked with E. B.
c^2 (512)	BC	N.H.	N.H.	85 s.u.	80 s.u.
	ABC	55 s.u.	N.H.	50 s.u.	55 s.u.
c^1 (256)	BC	75 s.u.	N.H.	75 s.u.	75 s.u.
	ABC	75 s.u.	N.H.	55 s.u.	55 s.u.

Tuning Fork Tests

c^2 (512)	AC	5 seconds		55 seconds	
	BC	10 seconds	N.H.	14 seconds	14 seconds
	ABC	25 seconds		30 seconds	

Weber — The Weber is referred to the right ear when the fork is on the midline of the vault or to the right side of the midline. To the left of the midline it is referred to the left ear. Masking the right ear does not change the responses. Blocking the right ear has no effect. Masking the left ear entirely eliminates the hearing for the fork. Blocking the left ear causes the fork to be referred to the left.

		RIGHT		*LEFT*	
Monochord	AC	14,000 d.v.	13,000 d.v.	16,000 d.v.	16,000 d.v.
	BC	15,000 d.v.	15,000 d.v.	18,000 d.v.	18,000 d.v.

Screening the left ear with the finger or masking the left ear with the electrical buzzer has had very little effect upon the hearing of the monochord in the right ear either for air or bone conduction. Masking the left ear with a strong current of air, on the other hand, eliminates all hearing in the right ear for bone or air conduction. The fact should be stressed that blocking the opposite ear with the finger has such little effect in preventing cross-hearing for the monochord.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Left Ear: Normal. Right Ear: Ethyl chloride elicits no response.

IMPRESSION: This case illustrates at least three important points. It is necessary to mask the good ear in the presence of unilateral deafness, both for air and bone conduction. There are great difficulties in masking the monochord. The Weber may be a very misleading test.

Using the masker in the right or deaf ear has very little effect upon the hearing for BC in the normal or left ear. This supports Bunch's (1931) statement of the effect of the masking tone on the better hearing ear.

Bone conduction tested on the audiometer for c^1 or c^2 in the deaf or right ear is within five sensation units of the hearing of the normal or left ear. Masking the left ear with a stream of air, or with the electrical buzzer, eliminates all hearing from the right ear for bone conduction. Furthermore, screening the good or left ear with the finger increases the apparent hearing in the deaf or right ear just as much as it does in the left or normal hearing ear. Screening the right ear, of course, has no effect upon the hearing. This suggests that the bone conduction is on the left rather than the right side.

CASE NO. 49 — Miss L. H., age 27. Admitted November 17, 1934.

DIAGNOSIS: Ependymoma of the third ventricle.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

November 17, 1934

NOSE and THROAT — Normal.

OTOSCOPY

Right Drum — Suggestion of a scar in the posterior half of the drum. No history of middle ear disease.

Left Drum — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass E_1 right and left.

BONE CONDUCTION TESTS

	RIGHT	*LEFT*
Tuning Fork Tests c^2 (512)		
Schwabach	Normal	Normal
Rinné	AC greater than BC	AC greater than BC
ABC	Normal	Normal

Weber — Not lateralized.

Monochord — Upper tone limit of 18,000 d.v. each ear.

Watch	15/20″	15/20″

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.
NOTE: The above examinations not done by the present investigators.

OPERATIVE PROCEDURES:

November 21, 1934 — Bilateral occipital trepanation and ventriculography.

November 21, 1934 — Operator, Dr. W. V. Cone. Myoplastic suboccipital craniotomy with partial removal of a cystic tumor of the posterior part of the 3rd ventricle. The removed cyst wall measured 3 cm. in diameter, and from 1 to 3 mm. in thickness.

May 13, 1936 — Good general condition, still some ataxia.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

May 13, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — (See Fig. 18).

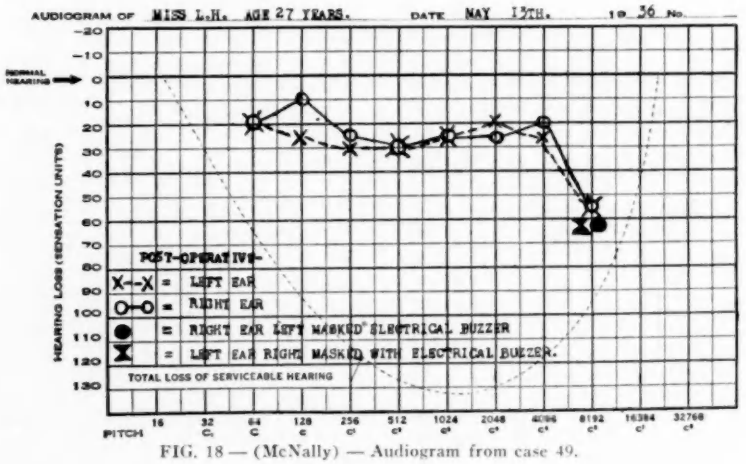


FIG. 18 — (McNally) — Audiogram from case 49.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	70 s.u.
	ABC	50 s.u.	45 s.u.
c^1 (256)	BC	55 s.u.	55 s.u.
	ABC	45 s.u.	40 s.u.

Tuning Fork Tests			
c^2 (512)	AC	55 seconds	55 seconds
	BC	20 seconds	20 seconds
	ABC	50 seconds	55 seconds

Weber — Not lateralized with BC or ABC.
NOTE: Test carried out in a very noisy ward.

Monochord	AC	13,000 d.v.	12,000 d.v.
	BC	15,000 d.v.	13,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: There is a definite drop post-operatively for the highest note of the audiometer, and it is bilateral. It is in part confirmed by the monochord. It is quite possible that this hearing defect may be in some way connected with the intracranial lesion.

CASE NO. 50 — Mr. L. K., age 36. Admitted March 6, 1936.

DIAGNOSIS: Neurofibroma, left orbital region.

PRELIMINARY OTOLARYNGOLOGICAL EXAMINATION

March 9, 1936

NOSE and THROAT — Normal.

OTOSCOPY — Normal.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2 bilateral.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	75 s.u.	80 s.u.
	ABC	60 s.u.	65 s.u.
c^1 (256)	BC	70 s.u.	75 s.u.
	ABC	55 s.u.	55 s.u.
Tuning Fork Tests			
c^2 (512)	AC	70 seconds	70 seconds
	BC	20 seconds	20 seconds
	ABC	35 seconds	35 seconds

Weber — Not lateralized for BC or ABC.

Monochord	AC	16,000 d.v.	16,000 d.v.
	BC	17,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

OPERATIVE PROCEDURE:

March 14, 1936 — Operator, Dr. W. V. Cone. Left frontal osteoplastic craniotomy with removal of neurofibroma within the muscle cone of the orbit. The measurements of this tumor were 2.6x2.4x2.2 cm., and its weight was 8.5 grams.

POST-OPERATIVE OTOLARYNGOLOGICAL EXAMINATION

March 25, 1936

NOSE and THROAT — Unchanged.

OTOSCOPY — Unchanged.

HEARING EXAMINATION

AUDIOGRAM — Subclass A_2, bilateral.

BONE CONDUCTION TESTS

Audiometer BC		*RIGHT*	*LEFT*
c^2 (512)	BC	80 s.u.	80 s.u.
	ABC	60 s.u.	60 s.u.
c^1 (256)	BC	75 s.u.	75 s.u.
	ABC	50 s.u.	50 s.u.

Tuning Fork Tests			
c^2 (512)	AC	75 seconds	75 seconds
	BC	20 seconds	20 seconds
	ABC	35 seconds	35 seconds
Weber — Not lateralized for BC or ABC.			
Monochord	AC	15,000 d.v.	16,000 d.v.
	BC	16,000 d.v.	17,000 d.v.

VESTIBULAR EXAMINATION — *Cold Caloric Test* — Normal.

IMPRESSION: A large osteoplastic craniotomy during which the dura was not incised has not caused any change in hearing.

SUMMARY OF GROUP SIX

In this group of five patients the lesions were as follows:

Tumor
- Basal Ganglia ..... Case 46
- Third Ventricle ..... Case 49

Bilateral Sub-dural Effusion ..... Case 47

Eighth Nerve Section — Right ..... Case 48

Osteoplastic Craniotomy ..... Case 50

In view of Dandy's findings in relation to mid-brain lesions, it is interesting to note that in the patient with the tumor of the basal ganglia, the hearing is normal. Following the removal of the third ventricle tumor there was some high tone loss which was not present before operation. If Dandy's observations about high tone loss are correct, a high tone loss might have been expected pre-operatively in each of these patients.

Detailed discussion of some of the patients in this group will be found under the following headings:

Cases 47 and 48 — Individual case report impressions
Case 49 — Gradual High Tone Loss

SUMMARY — ALL GROUPS

Of the fifty patients in this series, thirty-five had lesions involving the intracranial structures. Twenty-two of the patients showed some hearing loss. It was only in the four cases of eighth nerve tumor that there was a marked and characteristic loss of hearing which in each case was accompanied by changes in the

vestibular reactions. The slight hearing changes which occurred in some of the patients with lesions involving either the cerebrum, the cerebellum, the basal ganglia or the brain membranes, were not definitely typical of the lesion.

Apart from the four cases of eight nerve tumor, none of the remaining thirty-one cases of brain lesion showed any typical changes in bone conduction. In this series there were some atypical changes in bone conduction which we have attempted to explain in discussing the individual cases.

AUDIOGRAMS

In reviewing the cases it is noted that certain types of audiograms tend to recur throughout the different groups. All audiograms showing the same type of curves have been compared in the hope of discovering some common factor in their etiology.

Abrupt High Tone Loss — Dandy (1933 and 1934) states in the latter book, "If, therefore, the audiometer curves show an abrupt loss in the high tones on both sides (above 2048 vibrations) and a tumor is known to be present, I think it may be a localizing sign of importance, i.e., to the region of the midbrain." He illustrates such an audiogram in Fig. 60 on page 119 of his book. It is very similar to Figs. 6 and 10 of the present investigation. There are four such cases in which the audiograms are very much alike (cases 11, 17, 37 and 41).

Case 11 showed such a high tone loss only in the right ear. The case was one of posterior fossa tumor suspect. However, a ventriculography and an exploratory sub-occipital myoplastic craniotomy did not reveal any evidence of neoplasm. There was some evidence of old healed middle ear disease.

Case 17 showed a bilateral high tone loss more marked on the right side. The patient had suffered an extensive depressed fracture of the left frontal region and base of the skull. There was laceration of the left frontal lobe. The right sided ophthalmoplegia would suggest extensive damage in the right frontal basal region. There was bleeding from both ears at the time of the accident. Both vestibules reacted normally suggesting that the internal ears were normal. The hearing loss may have been due to the ear lesions at the time of the injury or to the intracranial lesions. The hearing loss on the audiometer was confirmed by the monochord but not by the bone conduction tests.

Case 37 shows a bilateral high tone loss partly confirmed by the monochord. A very large cyst had been removed from the left cerebellar lobe. There was no evidence of any local ear disease and such a large cyst, or its removal, may have caused some slight mid-brain damage (Dandy).

Case 41 shows a high tone loss in the left ear. It was detected after a large meningeal fibroblastoma was removed from the left cerebello-pontine angle without obvious damage to the 5th, 7th, and 8th nerves. The hearing loss is confirmed by the monochord. There is a history of old middle ear disease. The high tone loss may be due to the old middle ear disease, to slight operative trauma of the eighth nerve or to the effects of the large angle tumor so close to the mid-brain.

In three of the above four cases there is evidence of local ear disease. In two of the cases there is a possibility of traumatic nerve damage and in one case intracranial disease has not been proven. Only one of the cases has an uncomplicated intracranial lesion, a large unilateral cerebellar cyst.

Gradual High Tone Loss — In addition to the above cases showing an abrupt and marked high tone loss, there are six other cases (cases 18, 22, 26, 34, 35 and 49) showing a more gradual, or less marked, high tone loss, or more in keeping with Dandy's Fig. 33, page 77, and very similar to Fig. 18 of the present series.

Case 18 shows a slight high tone loss in the right ear. The case is one of skull fracture necessitating a sub-temporal decompression with removal of softened brain involving almost the whole of the right temporal lobe. At the time of the accident there was bleeding from the right ear. Either the ear damage or the brain injury may have caused the hearing loss.

Case 22 has a bilateral high tone loss on the audiometer partly confirmed by the fork and monochord tests. The hearing loss was noted after the removal of an area of brain tissue from immediately in front of the pre-central gyrus on the right side. It was for excision of a cerebral cicatrix. In case 20 the removal of a larger portion of brain tissue from this same area caused a bilateral dip in the audiogram at 4096 d.v., but the note 8192 d.v. was normal on each side (see also case 34). In all three cases the hearing lesions may be due to the brain tissue removal.

Case 26 shows on post-operative examination a right-sided high tone loss. A small cerebral cicatrix was removed from the left frontal adversive field near the pacchionian body. It is dif-

ficult to say that the hearing lesion is the result of this small cerebral lesion. The hearing loss is shown on the audiometer and on the monochord for air conduction. There is no change in bone conduction.

Case 34 shows only a very slight high tone loss on the left side. There is no change in bone conduction but the monochord is slightly down. However, the patient was over 50 years of age and some high tone loss is to be expected. The tumor in this case was very large and occupied the same area as in cases 20 and 22, the first of which showed a bilateral dip at 4096 d.v. and the latter showed a definite bilateral high tone loss. It is just possible that the tumor in this area or the operative approach to it interfered in some way with the nerve tracts for the high notes.

Case 35 shows a slight bilateral high tone loss partly confirmed by the monochord but not by the bone conduction tests. The patient was also over 50 years of age. The tumor removed in this case was fairly large and occupied the junction area of the occipital, parietal and temporal lobes, an area closely associated with cortical representation of hearing.

Case 49 which is a tumor of the third ventricle did not show any high tone loss on the audiometer just before operation. However, after the tumor was removed there was bilateral high tone loss (see Fig. 18). The hearing loss on the audiometer was confirmed by the monochord tests. It was in third ventricle tumors that Dandy most often found a bilateral high tone loss.

Of these last six cases showing some high tone hearing loss, one showed evidence of local ear injury and two others were over fifty years of age, all conditions which may explain some hearing loss. Two had lesions involving the temporal lobe, two had lesions just in front of the pre-central gyrus, one was a small lesion in the frontal adversive field near the pacchionian body and the sixth was a lesion of the third ventricle. The intracranial lesions cover a very wide area and it is difficult to imagine all these areas affecting high tone perception.

In addition to the audiometer high tone loss in the above ten cases, there is some loss of upper tone limit also shown on the monochord. In the most of the cases both air and bone conduction are never above 15,000 d.v. and only in case 11 does the upper limit go above 16,000 d.v. The high tone loss is never accompanied by changes in bone conduction when it is tested with the audiometer or tuning fork. This may not be surprising since the high

tone changes are slight and in a different pitch range from the audiometer and fork bone conduction range.

"Dip" at 4096 d.v. — In the last group of six cases the high tone loss is in the nature of a definite dip at 4096 d.v. with a return to normal limits at 8192 d.v. (see Fig. 9). Drury (1931) illustrates such an audiogram from a case of temporal lobe tumor. Ciocco (1932) considers such a dip in the audiogram at 4096 d.v. as possibly being "the first sign of a degenerative change in hearing which may continue until the hearing curve has reached the c^6 or c^8 stage" (see Guild's classification Figs. 3 and 4).

Case 20 shows a bilateral dip in the audiogram and there is a very slight lowering of the upper tone limit on the monochord. A large tumor was removed anterior to the left pre-central gyrus. This case is mentioned in connection with the discussion of cases 22 and 34, which had a similar brain lesion and showed definite high tone loss.

Case 29 shows a slight dip in the audiogram and some lowering of upper tone limit on the monochord. This patient had definite chronic rhinitis with possible eustachian tube changes giving some hearing loss. A very large meningeal fibroblastoma, bilateral, was removed from the fronto-parietal area. The tumor infiltrated the skull and pericranium and occluded the median longitudinal sinus over a distance of 10 cm.

Case 33 shows a bilateral dip in the audiogram and is illustrated in Fig. 9. A large meningeal fibroblastoma which completely filled the left middle fossa was removed. The patient had noted some hearing loss in the left ear for ten years but the tests did not show any special hearing loss on the left side. Such a very large tumor in the middle fossa may conceivably have caused some interference with nerve tracts concerned with high tone hearing. The removal of such a large tumor may have caused same slight injury to the mid-brain.

Case 36 shows a bilateral dip in the audiogram. A cyst the size of an orange was removed from the right cerebellar lobe. There was no evidence of local ear changes. Such a large cerebellar tumor could have caused some mid-brain damage.

In two of the last six cases, a diagnosis of an intracranial lesion was not confirmed. In one, the tumor was anterior to the pre-central gyrus and, as in two other similar cases, there was some high tone loss. In two other cases the tumor was found to be a

large meningeal fibroblastoma. In one of these the tumor was bilateral in the fronto-parietal area and in the other the tumor was unilateral and filled the middle fossa. The last case was a unilateral cerebellar cyst. Three of the four cases with definite intracranial lesions could conceivably have had some effect upon the brain stem. Four of the cases showed a slight lowering of the upper tone range for bone conduction on the monochord. A fifth case showed a slight lowering of air conduction on the monochord. There was no obvious change in bone conduction as tested by the audiometer or the fork.

Of these sixteen cases showing a high tone loss or dip in the region of the high tones in the audiogram, three were operated upon without any intracranial lesion being found. In six other cases the lesion was anterior to the pre-central gyrus. Of the seven remaining cases, three had temporal lobe lesions, three had cerebellar lesions and the last case had a lesion of the third ventricle. It is possible to imagine some interference with the mid-brain in the last mentioned four cases or even in the last seven cases, including the temporal lobe cases, but in the other six cases some central nervous system lesion, other than Dandy's mid-brain lesion would seem to be necessary to explain the high tone loss. Of course, as had been mentioned in connection with each case, there was a local ear lesion in some of the sixteen cases.

BONE CONDUCTION

It was stated at the outset that the object of the present investigation was to study bone conduction acuity in patients with proven inner ear or cochlear nerve lesions. Several methods of testing bone conduction were employed in the hope of determining whether one may be more suitable than the other. The results from each method have been tabulated and compared with one another but because it was impossible to demonstrate a consistent difference in the results obtained by the instruments used, these tables have not been reproduced.

There are slight variations between the results of the bone conduction whether it is tested by the audiometer, by the tuning fork or by the monochord, but they are not consistent nor are they very marked. Occasionally there is a change shown by one method which is not confirmed by any other part of the hearing test (see shortened BC and ABC ratio for audiometer BC at c^1 (256), case 27).

Bone conduction as tested by the audiometer was not found to be any more nor any less reliable than when tested by the tuning fork.

A comparison of the results of testing bone conduction on the audiometer at 512 d.v. with the results at 256 d.v. shows very little difference between the two. There are differences but they are rarely significant.

The records show that in the patients with other evidence of middle ear disease, the reading for BC and ABC, whether on the audiometer or with the tuning fork, more nearly approach each other (e.g., cases 8 and 13). It is also worthy of note that where there is this shortened ratio between BC and ABC there is frequently a tendency for lowering of the upper tone limit for both AC and BC on the monochord, another indication of high tone loss in connection with middle ear disease which has already been mentioned.

In surveying the whole fifty cases of this investigation one is impressed more by the similarity in the results of testing bone conduction by the different instruments than by any disparity. However, in testing certain problem cases, e.g., case 42, the bone conduction tests are not consistent and the results may not agree with the other tests of the examination. Such a case makes it very obvious that none of the present tests or none of the present methods are sufficient in themselves. It is only by doing both air and bone conduction tests by a variety of methods accompanied by suitable masking and by a vestibular examination that a true diagnosis may be reached.

Bone conduction tests with the monochord agree for the most part with the results on the audiometer and the tuning fork, particularly when the hearing lesion is bilateral. Monochord bone conduction is particularly misleading unless accompanied by masking and even then great care must be taken to make sure that the masking used is really effective (see case 48). This is still more important when the hearing lesion is unilateral. The electric buzzer which was used in this investigation was not always capable of masking the monochord BC from the non-tested ear. In these cases it was found necessary to use a strong current of air.

What has just been said for the monochord also applies for the fork and audiometer bone conduction tests. Bone conduction is so readily carried to the non-tested ear that in a case of marked unilateral ear disease a bone conduction test may be most misleading if not accompanied by masking (e.g., cases 42 and 48).

As in the cases just referred to, bone conduction may apparently be within normal limits in both ears even though one eighth nerve has been cut.

There have been a few very peculiar bone conduction reactions already mentioned that are difficult to explain. In case 48 the left eighth nerve had been destroyed and yet bone conduction tested by the audiometer and the fork was better on the side of the destroyed nerve. Masking the good ear with an electric buzzer increased the BC on the side of the destroyed nerve. The monochord BC which had been equal in both ears was increased in the damaged ear by masking the good ear.

Three patients with bilateral chronic suppurative otitis media (cases 5, 32 and 47) all showed evidence of advanced nerve deafness on one side and yet bone conduction on that side was equal to or better than bone conduction on the opposite or better hearing side. Masking was of some help in interpreting these results.

THE WEBER TEST

The Weber test was always performed with the fork c^2 (512). In eight cases it was referred to one or the other side when there was nothing in any of the other tests to support it. In four cases it was not referred to the side of the relatively increased BC. In two cases it was referred to the side on which the eighth nerve had been cut. In four cases it was properly lateralized according to the other tests. In the thirty-two remaining cases it was not lateralized. The Weber was therefore correctly referred in only four out of eighteen cases, and was decidedly misleading in the remaining fourteen cases.

THE MONOCHORD

The monochord proved to be of great value for testing the upper tone limit of air and bone conduction, particularly when used in conjunction with the 2A Western Electric Audiometer which does not go above 8196 d.v. Blocking the ear canal with the finger has very little effect upon the hearing for the monochord, whether by AC or BC.

Bunch (1928) points out that the monochord bone conduction is particularly valuable for testing the upper tone limit because the sounds are more intense than the high tones from other instruments and therefore will penetrate to the ear and give a truer estimate of the actual amount of nerve damage present in any one

case. He refers, of course, to the monochord accompanied by suitable masking.

VESTIBULAR EXAMINATION

Since the present investigation was concerned particularly with bone conduction tests, the vestibular examination was not done in detail in every case, but almost every patient had a caloric test done. If there was any suggestion of abnormality, additional vestibular tests were carried out.

It was only in some of the posterior fossa lesions involving the brain stem or eighth nerve that there was any change in the reactions. The supra-tentorial lesions did not alter the reactions.

In several cases where the diagnosis of the hearing lesion was in doubt the vestibular reactions were most important in finally solving the difficulty.

GENERAL SUMMARY AND CONCLUSIONS

In the present investigation, bone conduction was tested by the Western Electric audiometer bone conduction attachment, by the tuning fork, and by the monochord of von Struychen. One method did not prove any more reliable than the others.

The Hartmann tuning fork c^2 (512 d.v.) was found to be very suitable for carrying out the usual bone conduction tests. A standard method of activating the fork was used and was found to be a great advantage.

The 2A Western Electric Audiometer was used in all the examinations. The audiometer may be misleading if not kept in good repair by frequent checking.

When the 2A audiometer is used the monochord is particularly necessary for checking the upper tone limit of hearing.

The fork, the audiometer and the monochord tests must be accompanied by masking, whether the test is for air or bone conduction. It is particularly necessary where unilateral deafness is suspected.

The electric buzzer type of masker was found to be suitable in connection with the audiometer and fork tests, but to mask out the monochord it was occasionally necessary to use a strong current of air.

The absolute bone conduction test is considered to be a necessary part of any hearing test. It is particularly valuable when the

test is carried out under noisy conditions, as in the average hospital ward.

Much consideration was given to the range over which bone conduction should be tested. Evidence has been advanced to show that notes in the vicinity of 512 d.v. give the most reliable results. Bone conduction at 512 d.v. and at 256 d.v. was tested on the audiometer and the results are very similar throughout the series of fifty cases.

Increased bone conduction has been noted in some cases where there was obvious nerve deafness present or where the eighth nerve on the side of the increased bone conduction was known to be severed. No explanation is offered.

In a few cases, masking one ear caused an increase of bone conduction in the diseased ear. It is suggested that this may be due to the phenomenon of crossed paracusis.

Some evidence is advanced to show that encephalography and ventriculography cause intracranial pressure changes which last for twenty-four to forty-eight hours. In thirteen patients a complete hearing test was done immediately before and within twenty-four hours after one or another of the above procedures. The object was to determine the possible effect of intracranial pressure changes upon hearing. There was no effect upon the hearing in any of the patients examined. It must be pointed out that the amount of intracranial pressure change, if any did occur, was not measured in any of these cases so that any conclusions must be very guarded.

Following the removal of cerebral tissue in fourteen patients there were slight hearing changes in six. Most of the cases showed a slight loss of high tones on the audiometer and the monochord but the hearing loss can not be correlated with any special brain lesion.

Nine patients were examined from each of whom a cerebral tumor had been removed. Seven of the cases showed some loss of hearing but again the hearing lesion could not be associated with any special brain lesion.

Six patients were examined from each of whom a cerebellar tumor had been removed. Four of the cases showed some loss of high tones which in no case was typical of the cerebellar lesion.

The high tone loss noted in these patients with known brain lesions could not be distinguished from an early case of cochlear degeneration. Furthermore, the brain lesions which caused the

hearing loss were situated in many different areas of the cerebrum, cerebellum and basal ganglia.

In four cases of eighth nerve tumor there was complete nerve deafness on the side of the lesion but masking the normal ear is essential to proper interpretation of the tests. The vestibular reactions were absent on the side of the lesion and were of great value in making the diagnosis.

Thirty-five patients in the present series suffered from an intracranial lesion. In twenty-two there was some loss of hearing but in no case was the hearing loss typical of the brain lesion except in the cases of eighth nerve tumor.

The audiograms in these patients with high tone loss are of three different forms: an abrupt high tone loss in the region of 4096 d.v., a gradual high tone loss in this region, or a dip in the audiogram at 4096 d.v. The two first classes of audiograms are very similar to those which Dandy considers as suggestive of a mid-brain lesion. The dip at 4096 d.v. is considered by Ciocco to be indicative of early nerve degeneration.

The Weber test was carried out in most of the cases in this series and it has proven to be a very misleading test.

The most practical conclusion that follows from this investigation is not so much that any one test is better than another but that the more complete the examination the better. An accurate estimate of a patient's bone conduction can be made only after careful tests with the audiometer BC attachment, with tuning forks, and with the monochord have been carried out. These should be accompanied by air conduction tests on all three types of instruments. If the tests are not accompanied by suitable masking, both for air and bone conduction, they may be most misleading. A carefully taken history, a complete physical examination and the vestibular tests are often indispensable to a correct diagnosis of the ear condition.

Though the hearing in some of these patients may have been affected by the intracranial condition, the hearing tests have not proven of any value in localizing the intracranial lesion except in the cases of eighth nerve tumor.

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THE CLINICAL PROBLEM OF ALLERGY IN RELATION TO CONJUNCTIVITIS AND IRITIS*

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BALTIMORE

THE IMPORTANCE of allergy, or an altered state of reactivity to specific agents, has been stressed repeatedly in the recent ophthalmic literature. Much of this literature deals with purely experimental eye lesions and a small amount with clinical investigation. Rather broad deductions appear to have been made from these experimental and clinical findings, and at present clinical ophthalmologists are constantly confronted with the questions of therapeutic desensitization and various forms of vaccine treatment in patients with inflammatory lesions of the conjunctiva and anterior uveal tract.

I have no wish to minimize the importance of allergic reactions in these conditions. However, the questions involved are complicated, in many instances not clearly understood, and often lie more in the domain of the immunologist and bacteriologist than in the province of the ophthalmologist. It is my purpose in this paper to outline, from the viewpoint of the ophthalmologist, the present state of our knowledge on the relation of allergy to conjunctivitis and iritis, and to indicate the clinical application of this knowledge.

CONJUNCTIVITIS:

A large number of authors, Koutseff,[1] LaGrange,[2] Krückmann,[3] Berneaud,[4] Blake,[5] de Jean and Temple,[6] and numerous others, have reported cases of allergic conjunctivitis. Koutseff divides allergic conjunctivitis into two distinct clinical types, both due to direct contact of the sensitized conjunctiva with the specific allergin. The first is the sudden, edematous type, characterized by

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sudden onset, edema, conjunctival congestion and lacrimation. This he believes due to allergins suspended in the atmosphere, notably the pollens. The second is the follicular, eczematous type, coming on slowly, characterized by eczema of the lids and neighboring skin, and produced most frequently by drugs such as butyn, atropin and the various collyria and salves. LaGrange[2] describes three types: first, a transitory form, characterized by edema of the palpebral and bulbar conjunctiva, sudden in both its appearance and disappearance; second, the chronic blepharo-conjunctivitis, with a tendency to eczema of the lids; and, third, a chronic follicular type. LaGrange[2] believes the endocrine glands and the vegetative nervous system are concerned in allergic conjunctival disorders. Krückmann[3] likewise believes there are three types of allergic conjunctivitis. First, the sudden, edematous type, more or less strictly localized, with a glassy edema of the conjunctival fold without redness of the lid margins or evidence of furunculosis or hordeolum, sudden both in its appearance and disappearance; second, blepharo-conjunctivitis associated with eczema of the lids; and, third, a simple hyperemia lasting for weeks without much secretion of mucus. Krückmann believes vernal conjunctivitis occupies a special position. He also makes the point that the conjunctivitis appearing in hypersensitive individuals after the ingestion of certain foodstuffs, notably shell-fish and lentils, is indistinguishable from the conjunctivitis produced at times by atropin. This classification of three types of allergic conjunctivitis is excellent but the clinical appearance gives only little hint as to the nature of the causative allergin which may be pollen, animal dust, food, or bacterial toxins or their derivatives.

The best known example of the sudden edematous type of allergic conjunctivitis is that seen in asthma and hay-fever. The mucous membrane of the eye, the conjunctiva, participates in the general hypersensitivity of the mucous membranes of the upper respiratory passages, and like them reacts with a sudden inflammatory edema when in contact with the specific sensitizing substance, the air-suspended allergins. There is usually chemosis of the palpebral and bulbar conjunctiva, congestion, profuse lacrimation and a non-purulent secretion without bacteria. The attack subsides quickly after the instillation of adrenalin.

The classical example of the edematous, eczematous type of allergic conjunctivitis is that caused by drugs. This presents little diagnostic difficulty. The eczematous, cutaneous reaction and edema of the lids, the profuse lacrimation and conjunctival con-

gestion, usually with a little chemosis, present a characteristic picture. The offending drug can usually be identified without difficulty, for patients use few drugs in their eyes. This type of conjunctivitis is always accompanied by a marked generalized skin hypersensitivity which can be demonstrated by the ordinary patch test — the application of a salve containing the drug to the skin of the leg and covering this area with a bandage for twenty-four hours. If the patient is hypersensitive to the drug a marked dermatitis will occur at the site of application. By this test a suspected sensitivity can be determined before a drug is used. Occasional patients are sensitive to the oily base usually employed in salves. This type of conjunctivitis subsides slowly, in several days, after the withdrawal of the offending drug. A drug, entirely innocuous on first administration, may gradually acquire the properties of a sensitizing allergin. Landsteiner[7] showed that simple inert substances, even metals, may combine with native protein to form sensitizing substances. This is probably the explanation for the development of drug hypersensitivity by the conjunctiva. The offending drug, in contact with the lacrimal secretion, forms a foreign compound capable of producing the specific sensitization.

The third type of allergic conjunctivitis is the chronic recurrent, irritative conjunctivitis, often with a low grade folliculosis, with rather sharp exacerbations and consistently negative or normal bacteriologic findings. The responsible allergins may be pollens, animal dust, foods, or bacterial allergins. The detection and demonstration of the responsible allergins presents a difficult diagnostic problem. Our attention is drawn to such an etiologic possibility by the clinical picture, the normal character of the bacterial flora, and the resistance of the conjunctivitis to ordinary treatment, such as astringents, collyria, etc., and the correction of refractive errors and muscular anomalies. In this type of conjunctivitis the conjunctival reaction is often the only clinical evidence of tissue allergy.

The first step in the detection of the responsible allergin or allergins is a careful history. Are there other allergic manifestations, or a history of allergy in the patient or family, and is there any constant common factor in the patient's life and diet previous to the exacerbation? The second step is to test the patient against the specific individual and group allergins.

The best test for specific sensitivity is the intracutaneous injection of specific allergins. This is less misleading in its results and permits greater accuracy than does the scratch method. The

usual diagnostic dose of pollens, foods, and animal dusts is 1/40 of a cc. The diagnostic intracutaneous dose of bacterial toxins is 1/10 of a cc. of different dilutions. The reactions are read in fifteen minutes. Several tests can be done at one time. There are two distinct major groups of allergins against which the patient must be tested. First, the group comprising the pollens, foods, animal dusts, and a few specific allergins, and, second, the bacterial toxins.

The first general group of allergins, the pollens, foods, and animal dusts, consists of twelve sub-groups, the common pollens, the tree pollens, the rare pollens, epidermals and inhalants, vegetables, fruits, grains and spices, dairy products, meats, sea food, nuts, and special allergins, such as intestinal parasites, silk, rayon, etc. Each of these sub-groups contain from five to twenty-two allergins, in all about one hundred and forty different substances against which the patient must be individually tested, often in different dilutions. In addition to these twelve groups there are two other groups of composite antigens, made up of botanically related foods. In these two groups there are eighteen composite antigens, some containing as high as eight allergins. If a patient is sensitive to one of these group allergins he must be tested against each of the individual components of the group.

It is therefore quite apparent, when one considers the great number of allergins necessary to determine a suspected hypersensitivity and the complexity of the task involved, that such a study is beyond the province of the clinical ophthalmologist. The patient must be referred to an immunologist for the determination of his allergic status as regards the pollens, foods, animal dusts, and inhalants. When such a study has been completed the ophthalmologist is faced with the problem that the patient often shows a multiple sensitivity — is sensitive to a number of widely different allergins. There is also some evidence that skin sensitivity and conjunctival sensitivity may not be parallel. Frequently, therefore, it must be determined to which allergin, if any, there is an especial conjunctival sensitivity. This is determined by the ophthalmic test, the instillation into the conjunctival sac of a 1:10 or 1:100 dilution of the various allergins to which we have demonstrated a skin sensitivity. The eye should be quiet and the corneal epithelium intact, lest an allergic keratitis be produced. A positive ophthalmic test is evidenced by a prompt conjunctival congestion and lacrimation. If such a test is positive, the eye should be immediately irrigated and a drop of adrenalin instilled.

After a diagnosis of allergic conjunctivitis due to sensitivity to pollens, foods, or animal dusts, there arises the question of treatment. Theoretically, desensitization by repeated subcutaneous injections of the specific allergin is indicated. However, the length of time necessary to achieve such desensitization, the tendency of the hypersensitivity and symptomatology to recur, and the uncertainty of obtaining complete conjunctival desensitization are discouraging factors. We have not yet advanced sufficiently in our knowledge of ocular therapy to warrant the attempt at local desensitization by such methods as repeated local administration of the specific allergins. To my mind, the most important step is the education of the patient to avoid the specific intoxicating allergin. Thus a patient with a skin and conjunctival hypersensitivity to feathers must be taught to keep rigorously away from all poultry and birds and to sleep on a kapok pillow; a patient with food allergy must be cautioned to abstain from the specific food. The change in the clinical picture of the eyes, after following such simple advice, is often astounding. The only local treatment indicated is cleansing the eyes with any bland collyrium or salt solution, and the use of weak astringents with a little adrenalin. Until it is demonstrated that such expectant treatment is impractical or without avail, I do not feel justified in undertaking specific desensitization.

Bacterial Toxins: *A. Staphylococcus.*

The second large group of allergins are the bacterial toxins and nucleo-proteins. The most important of these, from the standpoint of the ophthalmologist, is staphylococcus toxin. With a small amount of training, an abnormal hypersensitivity to staphylococcus toxin can be determined by the ophthalmologist himself. Studies with other toxins, and with bacterial proteins, lie in the domain of the bacteriologist.

While staphylococcus toxin is lethal in small doses, it is not in itself irritative to a normal skin or mucous membrane. It produces irritative symptoms only in hypersensitive tissues. The probable mechanism of a conjunctivitis due to staphylococcus toxin may be conceived as follows: infection with a toxin-producing staphylococcus is followed by local absorption of the toxin and resultant tissue hypersensitivity. Fluctuation in the growth of the infecting bacteria is accompanied by changes in the toxin formation. The absorption of toxin by the hypersensitive conjunctiva results in an allergic inflammatory reaction, which in its

very nature is a chronic process, with periods of remission and exacerbation corresponding to the fluctuations in toxin formation and the exhaustion and recurrence of the local hypersensitivity. Even if the infecting organism is eradicated from the conjunctival sac, the tissue hypersensitivity to toxin remains and fresh infection with the same organism, which must be a common occurrence, is followed by a recurrence of the conjunctivitis. Hypersensitivity to the specific protein of the bacterial bodies of staphylococci is probably unimportant in the etiology of an allergic conjunctivitis.

Burky[8] has studied extensively the staphylococcus infections of the conjunctiva. He found that the staphylococci isolated from the normal and pathologic conjunctiva are of three different types. The first is an ordinary non-pathogenic albus type, occurring in the normal eye, and producing no symptoms. The second is a pus-producing, pigmented organism found in purulent conjunctivitis, furuncles, and pustular blepharitis. The third organism, termed "Ha" by Burky, is a hemolytic toxic-producing staphylococcus. This organism produces a strongly potent toxin, 0.1 cc. intravenously per kilogram of body weight being the lethal dose for an adult rabbit in twenty-four hours. Studies on human beings and animals by Burky and others have shown that young individuals are not skin sensitive to this toxin. As humans and animals grow older, however, and are exposed to constant and repeated infection with staphylococci, a cutaneous sensitivity of greater or lesser degree to this toxin may gradually develop. Likewise, substances develop in the blood which give a positive complement fixation reaction against an antigen made from this organism. Studies on humans and animals have indicated that a positive complement fixation reaction and a low degree of cutaneous sensitivity to the toxin indicate a comparatively high degree of immunity to the toxin and to staphylococcus infections, while a negative or weak complement fixation reaction with a high degree of cutaneous sensitivity indicate marked susceptibility. An individual or an experimental animal may be protected against the action of this toxin, actively by repeated intracutaneous injection of the toxin, this accomplishing desensitization or immunization; or passively by the administration of a prepared serum containing the specific staphylococcus antitoxin. Fundamentally important is the fact that the protection resulting from such active or passive immunization is potent not only against the specific toxin but against all staphylococcus infections.

Clinically, there is a large group of patients with recurrent, chronic conjunctivitis and marginal blepharitis in whom this susceptibility to staphylococcus toxin is apparently the specific causative factor. Practically, how may such a condition be detected and treated?

A presumptive diagnosis of an allergic conjunctivitis due to hyper sensitivity to staphylococcus toxin is made on the criteria of a chronic, recurrent, non-purulent conjunctivitis, often associated with folliculosis, marginal blepharitis, reddened, swollen lid margins, frequently slight secondary corneal changes, the elimination of other obvious causes for the conjunctivitis, the isolation of a toxin-producing staphylococcus from the conjunctiva, the presence of a high degree of cutaneous sensitivity to staphylococcus toxin, and usually a low degree of complement fixation against the staphylococcus antigen. Conjunctival cultures, taken after the omission of antiseptics, or during exacerbations of the inflammation, usually show the toxin-producing organism, although sometimes with all the other criteria for diagnosis present, the cultures are persistently negative, the organism itself presumably having been eradicated, but leaving the local hypersensitivity.

The cutaneous sensitivity is determined first by the intracutaneous injection of 0.1 cc. of a 1:100 dilution of the toxin, and later by the injection of 1:1000 or a 1:10 dilution, depending on whether the first injection gives a positive or a negative result. A normal individual reacts to the 1:100 dilution with an area of erythema approximately 3 to 4 cm. in diameter. Reactions beyond this, or to higher dilutions, are regarded as indicating an undue hypersensitivity. A strongly positive reaction is sometimes accompanied by a little local necrosis at the site of the injection. The complement fixation reaction is done by the usual technique, using as antigens a 1-8 dilution of the staphylococcus toxin, a 1:8 dilution of the filtrate of broth cultures of a pus-producing staphylococcus, and as a control a 1:8 dilution of the filtrate of the non-pathogenic organism.

The indicated treatment of such a conjunctivitis is active immunization or desensitization with the staphylococcus toxin. According to the degree of cutaneous sensitivity present, a dilution of the toxin is selected. This is usually 1:100, but in the extremely hypersensitive with reactions over 8x8 cm., a 1:1000 or a 1:10,000 dilution is used. All injections are given intracutaneously. The initial dose is 0.1 cc. This dose is repeated every four days until the local reaction becomes minimal. Usually from four to ten

injections are needed to reach this point. When a minimal local reaction to the first dilution is reached, the toxin dilution is then concentrated ten times, from a 1:1000 to a 1:100, or from a 1:100 to a 1:10 dilution, and 0.1 cc. of this stronger dilution is given until again a minimal reaction is obtained. This is continued until the patient can tolerate 0.1 cc. of straight toxin with a minimal reaction. This is apparently the point of desensitization. During such treatment occasional variations are sometimes seen, a patient on one injection reacting violently, while on subsequent injections there is only a minimal reaction, focal reactions in the inflammation coming on a few hours after the injection of the toxin and lasting twenty-four to thirty-six hours.

In the last few years in the Wilmer Institute over fifty patients with such an allergic conjunctivitis due to staphylococcus toxin have been treated. The results are difficult to tabulate on account of the varying degrees of sensitivity and ocular involvement, and the difficulty of achieving and maintaining desensitization. In general, the statement can be made that the therapeutic results have been surprisingly good, and that this form of treatment has offered, in otherwise hopeless and intractable cases, a real therapeutic weapon often giving spectacularly brilliant results.

In several patients passive immunization has been used — the injection of an antitoxic serum intramuscularly. This has been used in patients with a staphylococcic septicemia and in one blind cataractous individual with an allergic blepharo-conjunctivitis from staphylococcus toxin. In this patient one eye had been operated on elsewhere and lost from a post-operative infection. Local treatment to eradicate the staphylococcus from the conjunctiva was totally unavailing. This patient was finally given 20 cc. of a strong antitoxic rabbit's serum intramuscularly and was operated on twelve hours later. He received further daily injections of the antitoxin for five days until signs of a general reaction occurred when such treatment was stopped. Recovery from the operation was uneventful, without reaction or infection, although the conjunctival sac constantly carried a virulent toxin-producing staphylococcus.

There is one caution to be emphasized in the use of any foreign antiserum in patients with disease of the external eye. Severe reaction may be accompanied by marked edema of the lids. If there is corneal involvement, or such is feared, this sealing

of the eye by edema of the lids may be hazardous. For this reason passive immunization is not recommended unless the circumstances are such that there is no other course open.

BACTERIAL TOXINS: *B. Streptococcus.*

There is little actual information on the importance of the toxins of streptococci or other organisms in the production of an allergic conjunctivitis. Streptococci produce certain known toxins, hemolysins, leucocytin, scarlatina, and erysipelas toxins. Actual allergy to such toxins is a possibility, and it is conceivable that allergic inflammations of the external eye might result from such sensitization. Scholtz[9] has reported one case of conjunctivitis which he believed was clearly due to allergy to streptococcus toxin. However, the whole question of hypersensitivity to streptococci and their toxins is so complicated that it is impossible to speak with any certainty on the subject.

Primarily, there are two types of sensitivity to streptococci and their products. The first is the sensitivity shown to the intracutaneous injection of suspensions of the actual killed organisms. Such reactions, when positive, are of the delayed tuberculin type, coming on after twenty-four hours, and are probably indicative of an actual sensitivity to the nucleo-protein of the organisms. The second is the immediate cutaneous sensitivity shown to the intracutaneous injection of filtrates of these organisms, which may indicate a sensitivity to specific toxins, to the specific carbohydrate, to the products of bacterial growth, or even to substances in the culture media. A great percentage of entirely normal individuals show positive cutaneous reactions both to suspensions of the bacterial bodies and to the filtrates. Thus MacKenzie and Hanger[10] believed that skin hypersensitiveness to such bacterial substances was in general a function of age, or an index of normal exposure to infection, rather than an index of susceptibility to any specific substance or disease. Numerous other investigators have studied the problem and there is some evidence that the hypersensitivity is greater and more frequent in certain diseases, notably rheumatoid arthritis, supposedly related to focal infection.

The diagnostic problem is further complicated by the multiplicity of strains of hemolytic streptococci, each of which must be studied as an individual entity. Of the various groups of hemolytic streptococci pathogenic for man, group A contains over

thirty strains, group B contains three strains, and Group F contains four strains; Swift, Luncefield and Gardner[11] do not believe that different specific strains cause different disease conditions, but that the reaction to infection with any strain may vary from generalized infections to a simple pharyngitis, according to the individual reaction of the infected patient. The magnitude of detecting any specific sensitivity responsible for ocular inflammation can be realized when we consider the high percentage of non-specific reactions and the number of strains to be tested against.

In actual practice, the detection of a streptococcus in the bacterial flora of a chronic conjunctivitis might bring up the question of hypersensitivity to the bacterial protein or the products of bacterial growth being responsible for the local inflammation. Since there is no streptococcus toxin or vaccine which gives blanket protection, the only practical method of attacking the problem is the preparation of a killed suspension and of filtrates of cultures of the organism isolated from the patient, and testing the patient and controls against such antigens. If the patient shows an undue reaction to either the killed organism or to the filtrates, over that observed in the controls, it might be interpreted that hypersensitivity to this organism may be concerned in the clinical picture.

Inasmuch as we have no knowledge of any protective effect from the injection of a streptococcus toxin, the only treatment of a supposed allergic conjunctivitis caused by hypersensitivity to a specific streptococcus is vaccine therapy with the killed organisms. Swift and his co-workers[12] have shown that while intracutaneous injections of killed streptococci tend to sensitize rabbits, intravenous injection of the organism produces an immunity. In the treatment of hypersensitive rheumatoid arthritis patients, Wainwright[13] employed with success the intravenous injection of a salt solution suspension of the heat-killed organisms. An initial dose of 0.5 cc. containing approximately five million heat-killed organisms was given. The future doses were judged by the amount of reaction to the first dose. In general, the injections were given at four day intervals, increasing 0.5 cc. at each injection, keeping the dose below the point where general symptoms were produced. The patients were treated for periods of from two months to one year. If one must use a streptococcus vaccine, this appears to be the logical procedure.

VERNAL CONJUNCTIVITIS:

Vernal conjunctivitis is generally believed to be an allergic disturbance. The reasons for this belief are the seasonal occurrence of the attacks, the comparative absence of bacteria in the secretions, the tendency of the disease to attack the young, the specific hypersensitivity and general allergic tendency of vernal conjunctivitis patients, and the presence of eosinophiles in the conjunctival secretion. While an eosinophilia is not pathognomonic of an underlying allergy, it is either dependent on or closely related to protein absorption, and is usually found in allergic subjects.

The assumption that an underlying allergy is responsible for vernal catarrh is strengthened by the experimental demonstration that the conjunctiva is susceptible of specific sensitization and by the histologic study of the follicles on the bulbar and palpebral conjunctiva. The follicle which appears on the eye near the limbus has the general histologic picture of a phlyctenule, which is rather definitely proven to be an allergic manifestation. The second type of follicle, appearing on the palpebral conjunctiva, consists of papillary swelling with proliferation of connective tissue, infiltrated with lymphocytes, plasma cells, and eosinophiles. This reaction is somewhat comparable to that seen in the bronchial walls in asthma and in the skin in eczema, and has been spoken of as an allergic reaction characterized by persistent granulation.

In 1933 LaGrange and Delthil[14] published a short monograph on conjunctivitis and vernal catarrh and in the last three years have published other papers[2, 15] on the same subject. These authors definitely regard vernal conjunctivitis as an allergic disease of the pseudo-follicular type of conjunctival allergy. Their conclusion is based principally on clinical observation and study. They accept the specific sensitization of the conjunctiva as a proven experimental fact. The classical allergic syndrome of vernal conjunctivitis is: (1) the clinical picture, the recurrences, and the associated itching; (2) the climatic and seasonal incidence; (3) the frequent association of vernal conjunctivitis with other allergic conditions such as asthma, hay-fever, urticaria, angio-neurotic edema, etc.; (4) the absence of bacteria and inclusion bodies and the presence of eosinophiles in the conjunctival secretions; (5) the cutaneous sensitivity and the positive Prausnitz-Kustner reaction to the specific allergin shown by vernal conjunctivitis patients; (6) the reaction of the affected conjunctiva first to adrenalin and second to the specific allergin; (7) the frequent association of

endocrine disturbances and an imbalance of the vago-sympathetic system.

LaGrange and Delthil[14] are firmly of the opinion that other factors influence the allergic diathesis of the affected individuals. These factors are heredity and endocrine disturbances. They believe disorders of the endocrine glands induce a secondary imbalance of the vago-sympathetic system and that this predisposes to sensitization of the tissues and the resultant allergic reactions. They believe the conjunctival localization of the sensitivity and allergic reaction in vernal conjunctivitis is only another manifestation of the elective localization of cutaneous allergy, and possibly the direct exposure of the conjunctiva to direct sensitization may be responsible for this.

In support of these views these authors present in detail a large number of specimen clinical cases of allergic and vernal conjunctivitis, illustrating the various points emphasized in their allergic syndrome, especially endocrine disorders, affecting principally the sex glands, alterations in the oculo-cardiac reflex, and certain rather startling results obtained by organotherapy with various endocrine preparations. Especially interesting is LaGrange's last observation in 1935, which indicates that vernal catarrh patients show evidences of the vascular shock reaction, termed by Widal the "phénomènes colloidoclasique," seen constantly in experimental anaphylactic shock.

This is the evidence so far amassed that vernal conjunctivitis is an allergic disease — the clinical picture, the absence of bacteria, the seasonal occurrence, the experimental demonstration that the conjunctiva is susceptible of specific sensitization, the evidence that patients with the disease have a much higher percentage of associated allergy than do normal patients, the demonstration of a pathologic hypersensitivity to allergins in the greater percentage of such patients, and the pathologic picture of the diseased conjunctiva.

From the viewpoint of the clinician, how must such an assumption influence our approach to the study and treatment of this disease? The problem is little different from that already outlined for other forms of allergic conjunctivitis. The essential thing is to find the specific allergin or allergins to which the patient is hypersensitive and endeavor first to isolate the patient from these substances; and, second, since this is a recurrent, chronic disease, to achieve and maintain desensitization. Such procedures are no contraindication to other routine therapy,

astringent collyria, radium, or even organotherapy. Personally, I do not believe that our knowledge of endocrinopathies and endocrine therapy has yet progressed to the point where organotherapy is an indicated procedure except to a very limited degree.

Allergy and Focal Infection

Recent experimental work has indicated that certain types of recurrent uveitis and iritis may be an allergic reaction dependent upon sensitization and intoxication of the uveal tissue by bacterial derivatives. In 1933[16] I summed up the evidence in favor of this proposition. This idea was primarily suggested by the frequency with which sterile cultures were obtained from the iris and aqueous in acute iritis and by the flare-ups in the ocular inflammation which frequently followed the subcutaneous administration of bacterial vaccines. Experimentally this hypothesis found strong confirmation in the work of Swift and Derick,[17] Schultz,[18] Julianelle,[19] Seegal and Seegal,[20] and others. These investigators had shown that repeated intracutaneous injections of living streptococci or the absorption of bacterial products from an agar focus implant produced in the experimental animal a definite hypersensitivity of the ocular tissues, and later contact of the sensitized eye with the specific bacteria or bacterial products resulted in ocular inflammation. This was termed "the ophthalmic reaction." Since 1933 the experimental foundation for this theory has been still further strengthened. Seegal, Seegal and Khorazo[21] showed that if eyes sensitized by the direct injection of a specific protein in the anterior chamber were allowed to rest until all traces of specific protein had disappeared from the anterior chamber, and were then activated by an intravenous injection, traces of the specific antigen again appeared in the anterior chamber. This finding gave an explanation for the resensitization of eyes after the primary reaction, and readily explains why recurrent attacks of inflammation may take place. These same authors[22] also showed that if the eyes of experimental animals were subjected to some non-specific inflammation or insult at the time a foreign protein was circulating in the blood stream, the circulating foreign protein was absorbed directly into the eye and sensitization of the eye resulted. Julianelle, Morris, and Harrison[23] found certain cases of vascularization of the cornea to be an allergic phenomenon. Eyes sensitized by repeated intracutaneous and subconjunctival injections and later traumatized and exposed to the absorption of the specific antigens showed corneal vascularize-

tion. This they obtained with certain biological antigens, with egg albumen and with the purified nucleo-protein of various organisms. Such vascularization was likewise obtained by the repeated instillation of living cultures on the lightly scarified cornea, appearing after the ninth exposure. This apparent allergic reaction appeared as a local manifestation and had no relation to the general antibody content of the blood serum or to the heightened reactivity of the skin to the specific allergins. In 1934 Brown[24] repeated the work of Swift, Derick, and their co-workers. His experiments indicated that the ocular sensitivity resulting from infected agar implants might occur as early as the first to third day. Further, eyes directly sensitized by killed streptococci could be activated by remote infected agar implants, indicating that bacterial products diffusing out from a chronic focus of infection could produce inflammatory intra-ocular reactions in sensitized intra-ocular tissues. A further step forward was made by experiments of MacLean[25] in the Wilmer Institute. MacLean confirmed the earlier work of Swift, Derick and their co-workers on the ophthalmic reaction, and succeeded in producing a transient inflammatory uveal reaction in the untraumatized eye through intracutaneous sensitization and intravenous intoxication with living streptococci. Brown[26] recently showed that multiple sensitization of experimental eyes could be obtained by the injection of different allergins, both bacterial and protein. He further showed that the injection of an excessive dose of one of these allergins resulted in the subsequent inhibition of sensitization by other allergins. If typhoid vaccine was used as an "interfering allergin" and the antibody content of the aqueous was artificially increased, the intoxicating effects of other allergins was inhibited. This offers a possible new explanation for the effect of non-specific protein therapy — that the mobilization of non-specific antibodies in the aqueous inhibits the intoxicating effects of other allergins to which the eye may be sensitive.

A number of observers, notably Julianelle,[27] Seegal, Heidelberger and Jost[28] have endeavored to determine to which portion of the bacterial antigen mosiac the ocular sensitivity was due. Julianelle showed that the sensitivity resulting from the intracutaneous injections was apparently due to the nucleo-protein of the bacterial body, and Seegal and Heidelberger, working on the formation of precipitins to the specific carbohydrate of streptococcus hemolyticus, likewise confirmed Julianelle's work, indicating that the sensitivity was chiefly due to the nucleo-protein

fraction and not to the specific carbohydrate. In 1929 Tillet and Francis,[29] working with lobar pneumonia patients, showed that the immediate edematous skin reactions to pneumococci were due to the specific carbohydrate, while the delayed tuberculin type of reaction was due to the nucleo-protein of the pneumococcus.

Berens and his co-workers,[30] from the clinical viewpoint, studied the skin reactions and agglutinin titre of patients with ocular diseases against various organisms isolated from the nose, infected teeth and tonsils of such patients. They believe that such serobacteriologic studies give information as to the identity of the organism responsible for the ocular lesion, and that such reactions are of decided value to the ophthalmologist studying diseases of the eye due to focal infection; and likewise might indicate the specific bacteria from which vaccines for therapeutic use should be prepared. Benedict[31] has called attention to the relationship between infection in the female pelvis and certain recurrent ocular diseases, and the beneficial effects obtained with autogenous vaccines. There are other reports, too numerous to mention, on the use of autogenous vaccines isolated from foci of infection in the treatment of recurrent ocular disease.

Thus it is evident that a rather substantial experimental background has been built up for the hypothesis that recurrent iritis or uveitis may be an actual allergic manifestation, due to sensitization and intoxication from bacterial allergins. Confronted with this experimental evidence that allergy may be concerned in chronic, recurrent uveitis, and with the clinical evidence of heightened skin reactivity to certain bacteria harbored in suspected foci of infection, what should be the position of the clinical ophthalmologist on the problem? We are constantly confronted with the suggestion that the patient be skin-tested against all organisms isolated from any focus of infection, or even from the gastrointestinal tract, and that organisms to which he may be skin-reactive be assembled into an autogenous vaccine for therapeutic use. Such vaccine therapy, based solely on skin-reactivity to bacteria, in the present state of our knowledge, is to my mind an unjustifiable and scientifically indefensible procedure. In the normal course of life, any average human being is bound to become hypersensitive and skin-reactive to a great host of common bacteria to which he is constantly exposed. All studies, such as those of Derick and Fulton,[32] MacKenzie and Hanger,[10] Birkaug,[33] and numerous others, have shown that the greater percentage, not only of diseased but of normal individuals, are skin sensitive

to common organisms, such as streptococci and their derivatives. If we accept such skin reactivity as a criterion for vaccine therapy, there is no disease in the whole gamut of human pathology which should not be so treated. The patient has no chance of escape. Further, we do not clearly know whether the vaccine should be prepared from the bacterial bodies or the filtrates. In the case of staphylococci, as already pointed out, all evidence indicates the maximum protective effect is obtained by a properly prepared toxic filtrate, while with streptococci the present indications are for the use of a vaccine prepared from the bacterial bodies. To determine the proper preparation of vaccine, each suspected organism must be studied separately; otherwise we are guilty of the rankest kind of shotgun therapy. In the present state of our knowledge there appears to be little justification for the use of a vaccine prepared from the intestinal flora. In the case of organisms isolated from a focus of infection, the only indication for the use of a vaccine would be that, first, there is some reason from the nature of the illness, and exclusion of other causal factors, to suspect one individual organism; and, second, that the affected individual shows a far more intense skin-reactivity to this organism than would normally be expected, or is shown by other normal controls. Only with these criteria do I believe the use of a vaccine is justified in a recurrent uveitis, and then certainly not until our first duty — the eradication and treatment of the primary focus of infection — has been accomplished. In short, while there is much experimental evidence to indicate that certain cases of recurrent uveitis may be due to bacterial allergy from a focus of infection, we are far from the clinical proof of such a proposition, and with our present limited knowledge there is little justification for the present promiscuous use of vaccine therapy in recurrent uveitis.

CONCLUSIONS:

1. Allergic conjunctivitis may appear as either: (1) a sudden transitory conjunctivitis, characterized by edema, chemosis, and conjunctival congestion and injection; (2) as a chronic blepharo-conjunctivitis with a tendency to eczema of the lids; or (3) as a chronic, recurrent conjunctivitis, often complicated by folliculosis and blepharitis.
2. Such conjunctivitides may be due to pollens, food, animal dusts, inhalants, or bacterial toxins and proteins. The diagnosis

is made on the demonstration of a specific skin hypersensitivity and may be confirmed by an ophthalmic test. Treatment consists of isolation of the patient from the offending allergin, and specific desensitization.

3. The present evidence indicates that vernal catarrh is an allergic disturbance.
4. While there is much experimental evidence indicating that recurrent iritis may sometimes be an allergic reaction due to bacterial hypersensitivity, it is not proven clinically, and there is little justification for vaccine therapy in this condition.

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DISCUSSION

Dr. Anderson Hilding, Duluth, Minn.: The state of our knowledge concerning the relation of allergy to disease has been very much confused in all branches of medicine during the past decade. We owe Dr. Woods a debt of gratitude for doing so much to clarify the situation as far as ophthalmology is concerned. His knowledge is by no means limited to the clinical aspect of conjunctivitis and iritis. Behind his present contribution is a vast amount of experience and study extending over many years and far beyond the field of ophthalmology. He has gone deeply into the complicated study of immunity. When listening to what he has to say, one wishes that he had not limited himself so sharply, but that instead he might have included the keratidides and something about sensitization to lens matter and ocular pigment.

We all have experienced the difficulty of making a specific diagnosis. According to my understanding, the difficulty at least partially lies in the fact that allergy itself is non-specific. The disease is not sensitization to certain materials but it is a constitutional state in which the organism is prone to develop sensitization to such materials. I have a distinct impression received from my own practice that the allergin to which the patient becomes sensitive is one which he uses frequently or to which he is greatly exposed. There come to mind patients sensitive to substances as follows: two grocerymen, to spices; numerous young women to cosmetics; a tuberculous patient, to milk (she had been drinking two quarts daily for years); two grain dealers, to grain dust; a patient so fond of cheese that she ate it four times a day, sensitive to her cheese; a pathologist to formalin.

Under these circumstances it is easy to understand that if there should be a change of allergins in the environment, there might also be a corresponding shift of sensitization. If a patient succeeds in avoiding one substance to which he has been found sensitive, that is no guarantee that he may not develop other sensitizations. In fact, he would be prone to do so. Added to this difficulty is the fact that sensitization is seldom limited to one allergin but is usually multiple.

One young asthmatic girl comes to mind, who was known to be sensitive to sixty or seventy different materials. Her diet, for instance, was made up of only a very few foods to which she showed no reaction on the skin or otherwise. She would feel fairly well on these foods for a few weeks and then she would finally become sensitive to most of them, meanwhile losing her sensitization to some which she had not eaten for a long time. A new diet list would then be made to include the latter. In other words, her sensitization shifted definitely from one set of foods to another.

As to the interpretation of skin tests, one must keep in mind that the skin is a separate organ when it comes to immunity and allergy. That a certain allergin calls forth a vigorous response in the skin is no proof that the nasal mucosa, for instance, is also equally sensitive. The conjunctiva and skin are closely related structures and yet their sensitization is by no means parallel.

With these facts in mind: that there are several hundred materials in our environment to which people become sensitive, that each allergic individual is usually sensitive to few or many depending upon the severity of his allergy, that the sensitization may shift from one set of allergins to another, that skin sensitization does not run parallel to that of other organs, is it any wonder then that we find difficulty in making specific diagnoses. It is not strange that the patient cannot always be rendered symptom free, and be assured that there will be no recurrence.

Contrary to Dr. Woods' experience, our results have been very discouraging when treatment consisted only in eliminating those substances to which the patient showed positive skin reactions: for instance, removing from his diet those things which gave a positive test. The only way in which we have gotten results was through the much more elaborate process of test diets like those suggested by Rowe. In our practice we find difficulty in keeping the patient under treatment until a logical conclusion is reached. His ocular symptoms, at least from the conjunctiva, are often comparatively mild and he feels that diagnosis and treatment should be correspondingly easy, which, of course, does not follow.

Dr. Woods several times refers to immunity and allergy together as though closely related. They seem to be very closely related, and I confess to much confusion in my own mind, theories of immunity notwithstanding. Why should repeated small doses of proteins over a long period of time sometimes cause a high degree of immunity and at other times the very opposite? Why should it be true, as Dr. Woods points out, that we become sensitive, even though we show no symptoms, to the toxins of staphylococcus and streptococcus as we are exposed to them year after year? Why should we not become highly resistant? The sensitization to streptococcus found in rheumatoid arthritis and to the toxins of tubercle bacilli is more easily comprehended because in this case the organism is sick and to some extent at least has given way before the infection.

It seems to me that our best hope for successful treatment and prevention of allergy lies with the immunologists who may some day adequately explain the true nature of allergy. The clinical solution then may be quite simple.

Dr. Woods has referred to the possible relation of allergy to endocrine disturbance. The literature contains a number of references to such a relation. I should like to ask Dr. Woods for his personal opinion. Does he think that an endocrine disturbance is the basic factor underlying this tendency to develop sensitization to protein material?

There is one other thing that I would like to have him discuss. During the last two years I lost two eyes after cataract operation, eyes which had been subject to recurrent iritis over a period of 20 years. The second one got along well for nine days and then began to develop iritis, as had the first one. In the struggle which followed, the lady became sensitive to the drugs which we used, atropine, cocain and lubrichondrin. The skin became sensitive to the cotton used in the dressing and eventually she had swelling and eczema over the whole half of the face that continued for months, and the eye was lost.

Another patient lost one eye, after operation elsewhere. He attained 20/40 vision a few weeks after operation and then suffered recurrence of iritis. Vision is now down to 1/200 and he wants the other eye operated.

If Dr. Woods doesn't mind, I should like to have him discuss a procedure in a case like that.

DR. ALAN WOODS, closing: There are several points brought out in the discussion. The first question concerns something that nobody can clearly understand. It is an amazing thing that we can take streptococci and by intracutaneous injection produce sensitivity and by an intravenous injection produce immunity.

It all goes back to the fundamental idea that our allergin in the substance of protein must go to the portal of entry. It goes back to the idea of our applied skin vaccines, and so forth, but I think it is the method of demonstration, the method of absorption that must govern the individual reaction of the patients in the development of the immunity over sensitivity.

The next question is on multiple sensitivity. When you get these tremendously allergic people, sometimes sensitive to 70 or 80 substances, there is no place to start. You come up against something in the allergic individuals before you begin. I do not mean to minimize in any way the importance of desensitization or subcutaneous injections. I do think, however, before we go into the question of subcutaneous desensitization it is wise to try the simple method of isolating a patient. If you are going in for desensitization of the patient you have to keep him desensitized. It has a tremendous influence over the patient's life. Frequently, you can obtain remarkably good results by simple isolation of the patient from the offending allergy.

Coming to the question of endocrine disturbances, I do not know much about that. You get into the higher flights of pathology. We know a certain thing about endocrine disturbances, it is true. I do not think the therapy would be to sensitize the sex hormones. I see no indication of it in the literature. The thing has not progressed to the point where it is of value. I think that 99 out of 100 of the endocrine products on the market are pretty nearly worthless. I have no great faith in them and no great hope for endocrine therapy in these cases outside, of course, the use of thyroid.

The next question that was brought up was on post-operative effects. I want to know certain things about that patient before I operate on his eye. I want to know his reaction to the common drug that I am going to use, his reaction to atropin and analgesics that I propose to use for my anesthesia. I want to know definitely about the question of the man's susceptibility to the proteins.

I personally do not believe it is germane to this subject, but I would like to mention a recent paper which appeared in the last number of the American Journal of Ophthalmology by Dr. Burky. It gave our experiences in desensitization.

We have not been speaking of the results obtained with desensitization of the allergic individuals by injection of protein by itself. By using the method of mixing the toxin and using a toxin lead mixture, using it as an antigen to step up the action, we have had particularly good results in the several cases that we have tried. That method, of course, is being used by Swift in his recent experiments in desensitization and immunization with certain specific allergins which we have not been able to desensitize with otherwise.

I would like to know the reaction of bacterial toxins and definite drugs as to the specific density of protein. I would like to go through a good deal of desensitization before, with those toxin mixtures, I operated on that individual.

PRIMARY SARCOMA OF THE UVEAL TRACT
AN ANALYSIS OF TWENTY-SEVEN CASES*

S. Hanford McKee, B.A., M.D.

MONTREAL

For practical clinical purposes, there are two kinds of primary intraocular tumours (1) sarcoma of the uveal tract, which occurs as a rule in adult life and destroys by metastasis; (2) retinoblastoma, found generally in infants, has a familial tendency, and destroys by local extension and also by metastasis. As a rarity one also finds metastatic carcinoma and metastatic sarcoma of the choroid. In the uveal tract sarcoma appears much oftener in the choroid than in the iris or ciliary body. Fuchs[1] puts 6% of the cases in the iris, 9% in the ciliary body, and 85% in the choroid.

Malignant melanomata are relatively rare. According to Fuchs[2] they occur in about 0.066% of all eye cases. Stallard[3] states that during 1925-1931 at Moorfields, sarcoma of the choroid was found in 1 of every 4000 patients attending the out-patient clinic. They occur mostly between the fortieth and sixtieth years, though in rare instances they have been found in children. Males and females are affected about equally. Rarely does this disease present itself in both eyes.

The earliest clear reference to intraocular sarcomata arose from a discussion upon the subject of pigmentation. Laennec[4] in 1819 first drew attention to this peculiarity as a means of classification and nomenclature. Lawrence[5] in 1845, Tavignot[6] in 1853, and others, regarding them as malignant, while Sichel[7] in 1851 distinguished between benign and malignant melanosis. Virchow[8] in 1863 distinguished between simple melanoma, melanosarcoma, and melanocarcinoma, and laid stress upon the fact that pigmentation was of secondary importance, the real test of malignancy being the histological structure of the growths. In 1868, von Graefe and Knapp[9] simultaneously published clear and accurate accounts

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of the clinical course of these tumours. In 1931, Callender[10] reported in detail the examination of 111 cases, which had been sent to the Washington Museum. He described four or perhaps five specific types into which he believed all primary malignant uveal neoplasms could be classified. In 1933, von Hippel[11] from the Göttingen Clinic reported a series of 118 cases. The evidence of metastases in this series was 38%, the majority occurring within five years. He believes this figure is too small and that 50% would be approximately correct. In 1934, Callender[12] reported on a new series of 196 cases, which included his previous 111. Last year Terry and Johns[13] of Boston gave a preliminary statistical study of the cases from the Pathological Laboratory of the Massachusetts Eye and Ear Infirmary.

Sarcoma of the choroid appears in two well known forms: Circumscribed and Diffuse. In the latter, the choroid is diffusely thickened in its entirety. This form is rare. The clinical course of sarcoma of the choroid may be divided into four stages:

(1) The first manifests itself by detachment of the retina at the site of the tumour. Patients may notice a disturbance in the visual field, and examination with the ophthalmoscope now shows at the source of the tumour that the vessels are engorged, the veins dilated, and the colour of the retina grey or greyish blue. The retinal detachment is generally sharply defined and one can often discern the dark tumour mass upon which the superficial tortuous vessels are visible. At this stage the differential diagnosis is most important, especially now when so much operative attention is being given to detachment of the retina. In patients between the fortieth to sixtieth years, spontaneous detachment of the retina in a non-myopic eye should be held in great suspicion.

(2) The second stage is called the glaucomatous or inflammatory, where, with increase in the size of the intraocular growth, tension within the globe is raised.

(3) The third stage is marked by rupture of the tumour through the eyeball.

(4) The fourth stage is that of metastasis when metastatic nodes usually appear, first in the liver, then in the lung and other organs.

These four stages can take from two to four years, and the first two are considerably the longer.

Sarcoma of the choroid belongs to the most malignant of eye diseases. The prognosis in a certain percentage is absolutely un-

favourable for the life of the patient, if the eye is not removed early. Even then, the prognosis is by no means to be regarded as perfectly favourable. Local recurrences in the orbit and metastasis also may develop after the removal of the eye. Sarcoma of the uveal tract is hence to be regarded as one of the most malignant of diseases and one which in very many cases ends in death.

The origin of the essential pigment of the eye-melanin may have an important pathological bearing on the nature of intraocular tumours. Bloch isolated from the embryo of the broad bean 3.4 dehydroxyphenylaline, a substance which he called "dopa" and showed that it was readily changed by this oxidase to melanin. When this substance is added to epidermal cells of skin in frozen formaline-fixed sections, granules of melanin are formed (the "dopa" reaction).

Melanin is closely allied to adrenaline, and it is probable that the two substances are derived from the same precursors and form alternative end products in metabolism. When the cells are stimulated to proliferate so that there is an increase of the oxidative ferment, a pigmented tumour is the result, or a generalized melanomatosis, as in Addison's disease. It would seem from clinical observations that in the eye melanogenesis is a function both of epiblastic and mesoblastic tissues, and recent chemical work seems to substantiate this deduction. Chemistry falls in line with clinical observations and so far as the eye is concerned it seems clear that pigmentation may be either ectodermal or mesodermal, so that malignant melanomata may be either carcinomatous or sarcomatous. In this respect Terry and Johns[13] state, "In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms called sarcomata. In fact Masson and others have presented strong evidence that is indicative of ectodermal origin."

The question of pigmentation is an intriguing one. Under normal conditions pigment in the body occurs in the rete malphighii of the skin, in the eye, in muscle and in fat. Pigment formed under pathological conditions may be derived from the blood (hæmatogenous), from bile (hepatogenous) may be elaborated by various cells after the analogy of the normal pigment (metabolic), or pigment may be introduced into the body from without by drugs as in tattooing or by inhalation (extraneous). Melanin occurs normally as the colouring matter of hair, choroid of eye, of the skin, and in the pigment matter of many lower animals.

Pathologically, melanin occurs chiefly as the result of an excessive production of this pigment by cells normally forming it, as in freckles, melanotic tumours and Addison's disease. The manner of this excessive production by melanotic tumours is by no means solved, nor can the production of melanin on normal melanin bearing tissue be considered as satisfactorily settled. Quoting Terry and Johns, there are four possible sources of pigment in uveal sarcomata:

(1) The true melanotic pigment, produced or laid down in and between the tumour cells.
(2) Normal chromatophore pigment of the uveal tract engulfed by the growth of the tumour.
(3) Pigment epithelium of the retina that either has been engulfed in the tumour or had grown into that tumour.
(4) Blood pigment from hæmorrhages or stagnant blood spaces that had been invaded or engulfed by the tumour.

In a number of the less pigmented tumours of our series, pigment was found more dense at the base (it is sometimes quite possible to note this basal collection of pigment with the ophthalmoscope). The age and nutrition of the parts may be more important factors favouring melanin production than the cell type of the tumour.

In 1931, Major Callender[10] described four or perhaps five specific types into which all primary malignant uveal neoplasms can be classified:

1. *The spindle-cell type.* Sheets, whorls, and other irregular arrangements of spindle-shaped cells with long oval nuclei. The ends of the cell appear to terminate in fibres, so that the cells resemble fibroblasts. There is no argentophile reticulum except that accompanying the nutrient vessels. These spindle-cell tumours can be separated into two divisions by the nuclear characteristics:

Sub-type A. The nucleus has a delicate reticular structure in which the nucleolar material is not well-defined. These tumours are usually fairly heavily pigmented.

Sub-type B. The cell has a sharply defined, deeply stained small round nucleolus, usually situated near the center of the nucleus in a rather coarse nuclear network, is usually lightly pigmented.

2. *The fascicular type.* The cells of this type are elongated, sometimes forming fibre-like structures but rounded and polygonal shapes are seen. The characteristic nucleus is oval, has a distinct nucleolus, and closely resembles the nucleus of spindle-cell sub-type B. The majority of the cells are arranged in columns or fasciculi, the long axis of the cell being at right angles to that of the column. The cells radiate about the centre of the column in a palisade arrangement, the center being a lymphatic or capillary blood vessel. Pigmentation is usually scanty.

3. *The epithelioid type.* Polygonal cells usually of relatively large size, though there is considerable variation in both size and shape. The nucleus is large, round or somewhat oval, and the nucleolus is distinct. This type varies greatly in the degree of pigmentation.

4. *The mixed cell type.* Irregular mixtures of spindle and epithelioid types of cells, with occasional areas of the fascicular type. These tumours are very heavily pigmented.

Terry[13] states that from the standpoint of immaturity of cell forms, one would consider the epithelioid type to be the most malignant, but Callender actually found the mixed to be the most malignant. In his report of 196 cases classified in the above five groups there were

36.64% of deaths in the mixed type
25.00% of deaths in the fascicular type
21.42% of deaths in the epithelioid type
only 6.81% of deaths in 44 cases of spindle-cell B
No deaths in 25 cases of spindle-cell sub-type A

Since the war period, 27 cases of primary sarcoma of the eye have come under our observation. Fifteen eyes were from males; 12 were from females. The age incidence was as follows:

20-39 years .....6 cases
40-49 years .....6 cases
50-59 years .....7 cases
60-69 years .....6 cases
70 and over .....2 cases

In 16 cases, there was a history of visual disturbance that brought the patients to consultation. The last 4 cases which only recently

came to the Out-door Clinic had consulted the optical departments of departmental stores, without, of course, much relief. In 4 cases, there was a definite history of previous injury to the eye by contusion. The clinical diagnosis was made in all cases except two where diagnosis was impossible because of cataract and glaucoma, which prevented a view of the interior of the globe. Separation of the retina is the most common complication of malignant melanoma and was observed in all our choroidal cases except the two just noted.

Orbital recurrence has been rarely noted and in our series was present in only one case. Terry and Johns suggest that the orbit may be an unfertile field for tumours, and that Tenon's capsule may hold the tumour in check long enough at least for metastasis already existing or metastasis from the orbital recurrence to bring fatal issue before local recurrence manifests itself.

The liver is definitely the most fertile field for metastasis, and while most metastases are blood borne, metastasis by the cervical glands does occur. We were fortunate in obtaining post-mortems in two cases, details of which will show you how extensive the metastasis generally is.

A.L., an adult male of 50 years, was sent in to hospital in February, 1924, with a diagnosis of detachment of the retina in the left eye, sarcoma of the choroid. The eye was enucleated. He was readmitted to the Medical Ward in March, 1925, and died the following April. The autopsy report in part was as follows: Melanotic metastasis in the dura mater, brain, occipital bone, clavicle, third rib, diaphragm, pericardium, peritoneum, mesenteric and mediastinal lymph nodes, epicardium, myocardium, liver, pancreas, left kidney, adrenals, bladder mucosa, great omentum, thyroid gland, eleventh thoracic vertebræ. The liver showed extensive metastasis and extended to the level of the anterior superior spine in the midcentre line.

J.M., aged 52 years, came to post-mortem in February last from sarcoma of the choroid in the left eye with metastasis to the abdominal wall and intestinal obstruction. There were found metastases to the hilus of the left lung, peritoneum, intestines, mesentery, liver, kidneys, adrenals, perinephritic tissue, pancreas, bladder, left femoral glands, and the subcutaneous tissue of the abdomen.

Miss C., aged 35 years, complained of visual disturbance. A detachment of the retina was found and enucleation advised. This was in 1929. The patient died from multiple metastasis in 1931.

Mr. P., aged 46 years, had a similar history, his eye was enucleated in 1928, he died in 1931.

In the first two cases the cell structure was classified epithelioid, the third fascicular, and the fourth mixed. Compare these with the following:

Mr. D., aged 62 years, had his eye enucleated in 1926, he is alive and well. Mr. D., aged 34 years, had his eye removed in 1927, he is alive and well. Miss N., aged 21 years, had her eye removed in 1928, she is alive and well. Mrs. U., 72 years, had her eye removed in 1932, she is still alive and well. Mrs. R., aged 64 years, had her eye removed in 1931, and is alive and well. The cell structure in all this second group of cases belongs to the spindle-cell sub-type A group.

In our series of 27 cases, there were 26 tumours in the choroid and 1 in the ciliary body. Pigment, graded 1 to 4, was present in every case in varying degree.

The cellular classification has been altered from time to time during prolonged study. At present it stands as follows:

Mixed cell type .....2 cases
Epithelioid cell type .....7 cases
Fascicular cell type ..... 4 cases
Spindle-cell sub-type A .....14 cases

This result means, according to Callender's last report, that in a little over 50% of our cases the prognosis should have been and is in fact excellent. Likewise in the other 50% the prognosis is bad. You may remember that in von Hippel's figures he suggested metastasis in 50% would be approximately correct.

Conclusions: "In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms called sarcomata. It is believed by some that they arise from the epithelial cells of the retina, whether dispersed in the choroid or as misplaced elements. A name should also indicate in these tumours some relation to melanotic pigment since there can be little doubt that every one of these tumours has a tendency to produce melanin."[13] This was present in each one of the 27 cases of our series. Terry and Johns have on these grounds suggested the term "malignant melanoma" in place of "uveal sarcoma."

They concluded from their study that freedom from metastasis for the usual five year period was not a sufficient interval in which to determine the malignancy of malignant melanoma. Other observers have noted metastasis as late as twenty-four years after enucleation. In one of our cases, death occurred from multiple metastasis nine years after enucleation.

Separation of the retina is the most common complication of malignant melanoma. It was observed in all our cases except two. The disturbance of vision thus caused was what brought 16 of our 27 cases to consultation. May I remind you that a serous detachment of the retina may be the primary symptom also in metastatic carcinoma of the choroid. I repeat, in a patient between the fortieth and sixtieth year, a spontaneous detachment of the retina in a non-myopic eye should be held in the greatest suspicion.

Malignant melanomata can be graded according to their cellular morphology, although the value of this grading can only be determined by the study of many more cases with an intense follow-up, extending over a comparatively long period. It is imperative, if we wish to correctly classify these tumours by the cell type, that serial sections of the whole globe be made. Otherwise the chance of error is great.

While the duration of observation in this small series has been insufficient to allow definite conclusions to be drawn, unquestionably the follow-up of these cases tends to show that the spindle-cell sub-type A is definitely the most benign of the Callender types. To date no case of spindle-cell sub-type A in his series has given rise to metastases, while the mixed cell and other types have given rise to a large percentage of metastases and death. This study leads us to believe that the Callender cellular classification gives us very definite information as regards prognosis in this dread malignant disease. Surely a wonderful step forward, though we must ever bear in mind that it is only a step.

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DISCUSSION

DR. T. L. TERRY, Boston: A very considerable amount of knowledge concerning primary uveal tumors has accumulated, so much that no one paper can touch on all points of the problem. It is, therefore, of no great moment that the author in his introduction did not mention that uveal malignant melanoma can destroy by extension as well as by metastasis. In two instances I have observed extension into the cranial cavity with hemianopsia of the other eye after enucleation of the tumor bearing eye. It is also of no great moment that the author did not mention angioma of the choroid as a type of primary intraocular tumor. Hill and Dart found such a tumor may simulate malignant melanoma clinically. After enucleation the blood spaces empty greatly and no tumor mass was seen grossly. Microscopic study revealed the true nature of the disease. I have only recently had a parallel case.

Primary tumors of the iris appear to be less malignant than those of the choroid and they should be regarded in a different category.

I wish to disagree with the essayist and the many others who insist in dividing the tumors into the four stages. Such a division is not only rather artificial but also extremely misleading. The team "stages" immediately brings to mind a picture of development and metamorphosis in a sequential order. It also tends to lend a false sense of security if the eye is enucleated when the tumor is small, the retina unseparated, glaucoma not present, and the eye unruptured. In the series reported by Johns and myself in 1935, Stage four (metastases) was reached in one instance even before the complete development of Stage one, i.e., before separation of the retina. Stage four (metastases) was reached before Stage two (glaucoma) in seven instances. And further, Stage four (metastases) was reached before the advent of Stage three (extension of the tumor outside of globe) in seventeen instances. It must be stressed that metastasis may occur very early even before retinal separation. We are never sure of cure by enucleation. The prognosis cannot be judged by either the size or the so-called stage of the tumor. Therefore, it is never safe to delay enucleation after the diagnosis is made.

An increasing number of observers are being converted to the opinion of Masson that all chromatophores are of ectodermal origin particularly in relation to nerve endings. Embryologists have known for a long time that ectodermal and mesodermal elements mingle freely in certain organs and tissues.

I do not want any misunderstanding about that statement. I do not intend to suggest in any way that there is a migration of cells from the outer layer of the optic cup into what is to become the choroid. This extension does not occur because these things have been formed before the pigment appears. These cells that we find in the choroid, although they have their origin apparently in mesodermal tissue, there are many strong points to indicate that they are definitely of ectodermal origin.

Abnormal pigment in the body may be indicated by an increase in the amount of Zondek's intermedin or melanophore hormone in the blood. Some four or five years ago Dr. Ferguson, in New York state — I don't know whether in New York City or not — thought that it would be a good plan to see if this intermedin was increased in the blood stream of people having melanotic tumors, knowing already that there is a great increase in the intermediary lobe of the pituitary, as Zondek suggests, and that there is an increase in pigmentation over the body. This sort of test was carried out in Boston. I do not believe it has been reported as yet. However, thorough laboratory studies have shown that such a test is not of diagnostic or prognostic value.

Although Johns and I recommend its usage, the term "malignant melanoma" is not original with us. In 1880 Virchow used the term. It has been used more recently in specific reference to tumors of the eye in the textbook of Ewing on *Neoplastic Diseases* and by Friedenwald in Penfield's *Cytology and Cellular Pathology of the Central Nervous System.*

The most striking feature of Callender's cellular classification is that not one single case of tumor death has occurred in spindle A type. Is this truly a benign melanoma of the choroid capable of destroying the eye and even invading the orbit? The differentiation of the other types may eventually give a fairly accurate method of grading malignancy. A study of the reticulum as stained by the silver stain of Wilder is of value. The Klein test as applied by von Hippel to the uveal malignant melanoma must still prove its value.

One other point mentioned by Dr. McKee is that of serial section of the entire tumor. That, theoretically, is fine, but practically if we make serial sections of the entire tumor and the tumor entirely fills the globe we will have thousands of sections, and it would be a very prolonged work to examine carefully every single one of these sections. The underlying thought is certainly correct and we can take samples at short intervals so that we can see something of the picture of the tumor throughout. It varies a good deal at different levels. It also reveals evidence of perforation through the globe.

When one analyzes the paper of the essayist one finds much of practical clinical information. It shows the importance of the following points, which I stress at every opportunity.

1. Suspect malignant melanoma in every case of separated retina.
2. Suspect malignant melanoma in every case of unilateral glaucoma.
3. Suspect malignant melanoma in every case of unilateral uveitis. In these tumors when we get a great deal of necrosis or a moderate amount of necrosis even, we are very apt to get a uveitis. Of course, it is unilateral.
4. Suspect malignant melanoma in every blind eye, especially when it is impossible to see the fundus. There are a number of people who advocate doing operations on blind eyes with glaucoma in order to reduce the

pain, make the eye comfortable so the patient can continue, but from our experience with 97 cases, some 40 instances of tumor were found after the eye was opened and it was not even suspected clinically. I have talked about this so much at our local institution that the house officers now put out on most every eye that is removed "possible sarcoma." Well, that may be something of a joke, but at least it is in their minds for clinical differentiation at the time.

5. And finally, remember that an intraocular operation may stimulate the tumor to grow more rapidly and become more malignant as well as be furnished a tract of decreased resistance through which it may grow. In fact, some observers state that the most common channel by which the tumor extends from the eye is along the tract of an operative wound.

The author is to be complimented for his paper and may I refer especially to the thoroughness in which he has followed his patients. Such follow-up work is difficult. Out of over three hundred of our patients five years or more after enucleation, Johns was able to follow successfully only ninety-four. This necessitated persistent effort at correspondence, examination of vital statistics and census information.

Sir John Herbert Parsons, London, Eng.: I would like, first of all, sir, to say how very much I agree with every word of the remarks that Dr. Terry just made, with one exception. That exception is one of practical academic interest and not, therefore, of great importance from a clinical point of view. I do not think that the factors which he stressed are extraordinarily important. He really stressed almost all of the most important points from the point of view of eliminating, so far as possible, the evil effects of such malignant tumors as these sarcomata.

I am not going to bore you with long remarks. But, there are just one or two things that occur to me. The ordinary and flat sarcomata, I think, are somewhat like the secondary carcinomata which definitely grow in between the planes of choroid tissue. I think they are among the lymph spaces of the choroid and point to the origin of the flat sarcomata which also have a sort of alveolar structure, and in all probability the flat sarcomata, which are very rare, and are endotheliomata growing in the tissue spaces.

If you have a detachment of the retina, I pointed out many years ago — and I have seen a good many cases which confirm it — that you may get a simple detachment almost invariably in the lower part of the eye associated with a much smaller detachment due to the growth. That is to say, there is a simple detachment below and very often in early sarcomata the detachment of the growth may not be very obvious and the case may be regarded as simply detachment when it is due to a malignant growth in another portion of the fundus.

In regard to pigmentation, one of the most remarkable discoveries which has been made of recent years is that regarding the dopa reaction and the whole question of the origin of malignancies in the body. That is extraordinarily interesting, but I think it has led to some results which are not altogether justifiable. The question has been treated mostly by dermatologists dealing with pigmented nevi. I think they have been led

astray from their observations. There are other people who have studied this subject in regard to the pigmentation being of ectodermal origin. That was a point that Dr. Terry brought out, if I understood his remarks.

To my mind the tremendous interval which exists between the development of pigment in retinal epithelium and the development of the epithelium in the choroid after the membrane is broken and is very well established is a very strong argument, indeed, against the origin of pigment invariably from ectodermal tissues.

As the next point, I was going to say a few words on malignancy. Of course, I am always very chary of making any statements due to one's own experience without controlling them by statistical results, because I have so often found myself wrong.

I have thought, for instance, that a thing is more common in men than it is in women, or in certain ages, or something of that sort and I found by getting a series of cases that those ideas are not borne out. We are awfully apt to be biased by a run of cases.

For instance, during my term as a curator and pathologist, we had a tremendous run of cases of the retina. If I had gone merely on my own observations I should have come to the conclusion that it was much more common than actually it is.

I do think that everybody will admit that sarcoma is extremely malignant. I think that the question of malignancy is rather a complicated one. These are points on which I agree with Dr. Terry. First of all, I agree with him as to the stages, getting away from the technical point of view.

I have always emphasized most strongly to the students that those stages are not chronological stages but that they may occur at different times. You do get the stages in most cases, but they are not chronological, as he pointed out; therefore, they may be misleading.

With regard to malignancy, there is first of all the question of pigmentation. I personally think that pigmentation has very little reference at all to malignancy in sarcoma of the choroid. Most of these tumors are pigmented, but you do get malignant true leukosarcoma. Some of the pigmented tumors are quite definitely really leukomata which have hematogenous pigmentation. They are usually probably the angioid type not in the ordinary sense of the word, but the vascular type of sarcoma. In those cases the pigmentation is a different type of pigmentation altogether. Even there, the hematogenous pigmentation may become iron-free and it would be impossible to distinguish it from the other. I do not believe the pigmentation has much to do with it and the fact of the growth being pigmented makes one extremely suspicious as to malignancy, unduly so. That is due to the fact that a vast majority of them are pigmented.

I doubt very much whether the action of pigmentation has very much to do with it. One gets an unpigmented body in the conjunctiva. You get them in the skin, too, as both of these types are liable and very relatively do become malignant. I do not think there is much to choose between one and the other.

The most important point about malignancy is the actual site of the tumor in the eye. That cuts two ways. In the first place, if you cut a tumor in its early stages you will probably have it in its early stages. If it is discovered, the eye will be enucleated at an early stage. Personally, I

think from my experience that those cases show a prognosis that is really quite good with regard to subsequent developments. But if the tumor is situated at the periphery it is less likely to be spotted as early as these central growths. Consequently, there is delay and very often very considerable delay before the eye is removed and the prognosis is much worse.

Many years ago there were some collected cases of sarcoma of the ciliary bodies, and we came to the conclusion that sarcoma of the ciliary bodies was less malignant than that of the choroid. From the other point of view they are absolutely identical. I think the conclusions as stated remind one that they might be due to the fact that he took a lot of other pupils' notes and presumed that these notes were equally reliable in different cases. Those things are very dangerous.

The other thing is that I think ciliary sarcoma is more or less likely to be spotted as an early case than sarcoma of the choroid and farther back than those cases in the ciliary region.

There is another point, if a growth is in immediate proximity, for instance, to the vortex vein, or some such position perforating the posterior ciliary vessels, and so on. In those cases it is natural to expect that dissemination may take place very early. But I do not think one ought to be too pessimistic about the actual malignancy of these growths. We must all of us have had experience with people who lived many years after they had melanosarcoma. The one which struck me more than any was a man whom I recommended to have his eye out for sarcoma of the choroid. I subsequently had to spend three-quarters of an hour in the middle of a busy morning trying to convince him that it was wiser to have his eye out that to undertake Christian Science treatment. Fortunately, I succeeded and he now plays a good game of golf in spite of the fact that the eye was removed.

In many cases the prognosis is quite good if you get the eye early. On the other hand, we want to admit that we are never certain about them.

THE CLINICAL IMPORTANCE OF PIGMENT IN THE FUNDUS

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THE PIGMENT of the fundus presents many interesting, complicated and obscure designs differing in distribution, intensity of color and layer location. At times the pattern is so distinctive, so common and so well recognized that even the immature ophthalmologist can draw accurate conclusions regarding its cause and duration.

In other cases the pigment display is so unusual, either because of the type of isolated pigment deposit or its site, that it calls for analysis by an expert. Actually, the variations are innumerable. The gradations in shade cover the entire color scale from the palest yellow to coal black. There are as many colors in the fundi as are found on a wooded hillside in the temperate zone in late summer. Pigmentations of the fundus depend upon the color of hair and the amount of pigment in the skin. Some types were classified and have been recognized since the earliest days of ophthalmoscopy as the albino, the blonde, the brunette and the negroid fundi.

Unusual pigmentation, that which is different from the normal, may be congenital, senile, the result of inflammation, new growth, trauma or degeneration.

The pathognomonic appearance of congenital pigmentation is that of irregularly rounded dark areas consisting of definite granules, the spots are smaller nearer the posterior pole, and larger toward the periphery. They lie beneath the retinal vessels. There is no marginal elevation for the substance of the deposit is of uniform thickness, and remains unchanged. They have no influence on vision, are most often dark or pale brown, although sometimes slate gray, and are discovered in the course of the routine examination.

Another type of congenital change should be subdivided into two groups, one the frank, unmistakable developmental absence, coloboma of the choroid, and the other intrauterine retinochoroiditis. In the former the heavily pigmented edge surrounding part or all of the depressed region is sharply delimited, while in the second, there is some question as to whether the sunken part should be considered an anatomic malformation or the result of changes, probably inflammatory. These latter include the borderline cases of so-called coloboma of the macula. It is very difficult to always exclude disease. In those which we call the inflammatory cases the appearance is often similar to that found in childhood or adult retinochoroiditis.

In true coloboma the edge of the lesion seems to be continuous with the surrounding retina. The lesion presents a sharp declivity, whereas in the other types the depression is shallow and seems to merge or fade into the retina. In the inflammatory forms certain other changes assist in the differentiation. The most characteristic of these are an increased visibility of Cloquet's canal, a delicate white film overlying some or all of the scarred region, and the dispersion of pigment over parts of the white region.

Senile pigmentation, best illustrated by the heavily pigmented drusen, may lead to confusion only when some of the clinical signs and symptoms are unrecognized. In such fundi there are always many rounded depigmented spots readily located beneath the retinal vessels. Often a macular choroiditis is engrafted on or takes place in an eye with drusen. The destroyed areas are then larger, the pigmented borders thicker and more evident. The functional tests aid in differentiation for the vision is reduced, whereas in the drusen no appreciable change is noted.

The pigmentation which follows the so-called senile macular degeneration, which is probably arteriosclerosis of the macular vessels, with secondary gross or minute hemorrhages, is always mottled gray to black, sieve-like in appearance but granular on accurate focusing and in red light found to be in the deeper retinal layers.

The pigmentations following retinal and choroidal inflammations comprise the largest group and present the most diverse forms. Some of these have been given the name of the author who first described them, others are classified by the portions of the fundus occupied and still others by their anatomic location. All have certain common characteristics. They are round or oval, single or multiple, discrete or confluent, all tend to clear in the

center while the pigment is deposited in and about the borders as gray, brown or black arcs, rings, masses or heaps. No two cases are exactly alike.

The condition called choroiditis juxtapapillaris is worthy of discussion. In some the patch clearly extends on to the nerve-head without sector field defects, while in others the disk is evidently separated from the region of choroidal destruction and yet there is a great sector loss of the field. It suggests the time has come for a more restricted use of the term. In other choroiditis cases the pigment layers may become matted together and lie over the sclera as black bridges over a milky lake, the retinal vessels coursing over the patch. One region may be outlined by a very narrow edge, while another may be heavily pigmented and the center a black plaque.

In tuberculosis the pigmentation is thinner and appears later, a fact of some clinical significance. In miliary or small isolated tubercles the lesion may be scarcely pigmented, but in the large, solitary tubercle the chronicity of the process leads to excessive pigmentation about the scar, outlining it with a heavy black ring.

In sympathetic ophthalmia, white and dark spots are frequently seen before the media becomes too dense for ophthalmoscopic study.

Pigmentation the result of syphilis may be in the form of small faintly pigmented spots. These are found in both congenital and acquired syphilis, in the former they give the picture called by the older writers pepper and salt fundus. In the acquired, the patches are larger with white centers and heavier marginal pigmentation. Other cases show extensive choroidal destruction with great white atrophic scars and much pigment. In other forms the pigment suggests that found in retinitis pigmentosa, but the subdivided deposits are more irregular, oval, oblong, granular or speck-like. In both there is nightblindness and later secondary optic atrophy.

When the pigment is in solution it frequently fills the lymphatic space about the blood vessels presenting an encircling black sheath or tube of unmistakable etiology.

The most destructive type is that which causes a disappearance of the choroid with an ever-decreasing pigmentation, the pigment collects as rounded masses without characteristic form or thickness, the fundus background is dirty, yellowish-white, the vessels are small and disk eventually becomes atrophic.

Syphilis can usually be distinguished from retinitis pigmentosa by serial stereoscopic fundus photographs and a careful clinical investigation. Retinitis pigmentosa may be present in a patient with acquired syphilis and under such circumstances the analytical ability of the ophthalmologist must be utilized.

When a new growth of the retina or choroid is pigmented the location and type may be of supreme importance for by them an earlier confirmation of the diagnosis is possible and very accurate landmarks are established for future measurements.

In some sarcomas of the choroid the dark brown mottling has served to label the detachment as one resulting from a tumor rather than following an injury or appearing spontaneously. The discovery, recognition and correct evaluation of pigment in malignancies of the fundus cannot be too often stressed.

Penetration of the globe is always attended by pigmentation, sometimes light in color and slight in amount, on other occasions great black sheets surround the scar as for instance after traumatic evulsion of the optic nerve.

In a contusion of the globe resulting in rupture of the choroid, the tears become black bordered, while in compressions an immediate edema of the retina is followed by depigmentation and innumerable black spots on a pale, dirty yellow gray background. The granules are usually separate and distributed somewhat uniformly over the involved area. This type of pigmentation follows retinal detachment operations, hole in the macula, tears in the retina and what is often considered a trivial blow. Critical inspection will enable the careful ophthalmologist to segregate these cases from the inflammatory or degenerative pigmentations for the deposits always lie in a discolored, disorganized part of the fundus.

The determination of the amount of trauma necessary to destroy the retina and the length of time which must elapse before pigmentation takes place must be investigated. In 1928, in the Archives of Ophthalmology, I placed on record a photographic history proving that the specking took place within a very few days.

The degenerations include retinitis pigmentosa, choroideremia and retinitis punctata albescens, all of which present many bizarre forms, some rare, all engaging.

The first visible evidence of retinitis pigmentosa is a deposit of pigment in or near an equatorial zone, this extends later both toward the disk and toward the periphery. It is not inflammatory,

but depends upon choroidal vessel closure which leads to the migration of the pigment into the well known bone corpuscle or spider-shaped collections. The extreme attenuation of the retinal vessels and the optic atrophy are subsequent to the equatorial region destruction. Incidentally, it might be well for some who claim improvement after therapy directed to the retinal vessels to investigate the basic conditions attending this disease before exploiting new cures or operations. Within the past few years attempts have been made to consider most of the cases of nightblindness as retinitis pigmentosa, and this has led to greater care in segregating retinitis albescens punctata and choroideremia.

Another peculiar, and as yet comparatively seldom recognized pigmentation, is that of the terminal stage of hypertension. It is of considerable prognostic value and deserves more attention than has been given to it in the past. The dark, chocolate brown spots are not of uniform size or definite location. Although they lie beneath the retinal vessels and are always discrete and distinctive they are always accompanied by the dominant signs of third or fourth stage retinal hypertension; that is, the edema has almost disappeared, the vessels are in a state of advanced arteriosclerosis, and hemorrhages may or may not be present. These spots are not an integral part of even the majority of the terminal stage hypertensions but when present they have an ominous significance, always forecasting death, for they appear only near the close of life. They should not be confused with hemorrhages or the retinal and choroidal scars found in the hypertension of pregnancy.

It seems advisable to restrict the use of the term retinitis punctata albescens to those cases in which there is nightblindness and a fundus studded with small white spots, irregular lace-like pigment deposits, small retinal vessels and optic atrophy. By such limitation the transitory post-inflammatory, post-traumatic or post-retinal edema white spots will be excluded and a more definite clinical syndrome established.

Choroideremia is so destructive and the dissolution of the pigment so characteristic that mention must be made of the pale or white fundus with streaks of pigment, small clumps or simply a brownish smudge, a definite clinical entity which will eventually be placed on a pathological basis.

Some pigmentations are not always distinctive for at times the area caused by trauma may be almost the same as a portion from a retinitis pigmentosa. A reexamination especially of photographs will probably always dispel any doubt, for in trauma the

spots are more granular and blacker than in retinitis pigmentosa. There are some that can only be diagnosed when all things such as presence or absence of nightblindness, injury or local infection, and the cause of the disease are considered.

A most peculiar and as yet baffling disease is angioid streaks, where the circumpapillary ring and the granular red linear extensions have not been examined by the pathologist. The picture is unique.

The black streaks and lines in certain fundi may be folds, but too few have been pathologically studied to be certain that all similar fundus appearances are caused by the same thing.

SUMMARY: Variations in the pigmentation of the fundus assist in the diagnosis of ocular diseases and are often a reflection of the condition of general health.

The fundus color depends upon the amount of pigment in the hair and skin.

Congenital deposits are of no pathological significance but may confuse the uninitiated. They are flat pigmented collections placed near the disk and peripherally.

Senile pigmentation is an exaggeration of drusen, Tay's choroiditis and senile macular degeneration. The benign drusen may become so large and so central as to embarrass the ophthalmologist who does not use the discriminating tests for vision and field defects.

Inflammations of the retina are always accompanied by the migration of pigment. The picture depends upon the number and size of the patch or patches. Many characteristic forms have been described during the eighty years of ophthalmoscopy.

When the retina is detached, the appearance of unusual pigment may be the determining factor in diagnosing an underlying malignancy.

All penetrating wounds of the sclera and all severe globe contusions leave pigment evidence of damage. The degree and site are often of great medico-legal value.

Of particular importance is a complete understanding of the pigmentations in syphilis and retinal hypertension.

Retinitis pigmentosa, retinitis punctata albescens and choroideremia should be separated and not grouped as evidence of a single cause until it has been convincingly demonstrated and proved.

CONCLUSION: The pigmentations of the fundus are distinctive, and when considered as one element in diagnosis, they are of supreme clinical importance.

* * *

Each phase of this subject was illustrated by lantern slides made from fundus photographs, some of which were in direct color.

DISCUSSION

DR. W. E. FRY, Philadelphia: Pigmentary changes in the fundus form striking and interesting figures for ophthalmological observation. You have just seen that they can be clearly shown in photographs. Probably in most instances the pigmentary change is not primary, but is secondary to another cause. That cause may be congenital, an acute or chronic inflammatory condition, malignancy, or be due to glaucoma or to senility. We might, therefore, say that the pigment picture seen ophthalmoscopically forms the fluorescent screen which allows us to observe and deduce changes which have occurred in the choroid and retina and which would otherwise not be easily noticed.

There are certain facts concerning intraocular pigment with which we are familiar, but which may be briefly mentioned at this time. It occurs as a melano protein. The pigment normally occurs in cells of three types, one is in two forms. It is in the rod or needle pigmentary layer of the retina, and in the choroid it is in the clump cells and chromatophores. In the latter two cells it is in the form of granules, while it frequently occurs in the form of short rods or needles. The pigment in the retina is not so dark as that in the choroid. There is probably no fundamental difference in the two types of pigment.

There has been some difference of opinion concerning the origin of the pigment. Some have considered that the pigment originates in the pigment epithelial cells and migrates from there into the choroid. Others have considered that the pigment originates separately in the retina and in the choroid. The latter opinion is probably correct. By means of the dopa reaction Black found that only epidermal cells gave a positive reaction and the mesodermal cells were negative. He therefore concluded that the pigment in the choroid resulted from a migration from the retina. However, embryologically it has been shown that the pigment appears very early in the retina, i.e., at the fifth week and very late in the choroid, and at the fifth to seventh month after the development of the lamina vitria, and that it appears first in the outer layers of the choroid. In the pigmented epithelium the pigment arises on the cytoplasms and is in the form of a melano protein.

Pathologically, pigment may appear in other cells, much as in fibrocytes, and in clasmatocytes of cells of the retino-endothelial system. Pigmentation of the former cells is always finer and less dense than in the latter cells.

In certain conditions, such as disturbed nutrition, the chromatophores which normally have branched processes lose their processes and have the appearance of clump cells. This has led various authors to believe that these two cells are variations of the same cell.

The pigment in the eye is not an inert substance but has certain irritative qualities and immunologically it is capable of acting as an antigen. The former ability is shown when pigment is injected intraocularly, when it produces a certain amount of retinal degeneration. The latter quality has been seen in relation to sympathetic ophthalmia.

Certain analogies have been made between dermatological and ophthalmic conditions. The retinal pigment epithelium has been compared to the epidermic and the uveal tract to the corium. A particularly important relationship has been shown between angioid streaks of the retina and pseudo-xanthoma elasticum. Each of these conditions in its field is rare and their association together is a relationship of great importance. There have been certain facts brought out concerning pigmentation in the skin and vitamin C and copper. There are skin conditions that have as one feature an increased pigmentation, namely, scurvy and Addison's disease. It has been found that the administration of vitamin C will prevent the hyperpigmentation shown in these diseases. An idea of the site of the reaction is obtained by the use of dopa. Vitamin C interferes with this reaction. In the formation of pigment apparently copper acts as a catalyst and it has been shown that a definite proportion exists between the amount of pigment, vitamin C, and copper present in the skin. In view of the fact that when pigment is introduced into the eye it causes a retinal degeneration, vitamin C therapy may be of use in the treatment of conditions characterized by retinal hyperpigmentation in spite of the fact that the pigment may be only a secondary factor.

Dr. T. L. Terry, Boston: No one questions the value of the ophthalmoscopic observations and photographic work of Dr. Bedell. The photograph gives a record of relatively two dimensions. In stereo-photographs binocular parallax must be small because of the size of the pupil and its distance from the retina, so small in fact that differences in level when slight could not be observed. I should like to ask Dr. Bedell if he has any definite evidence showing how small an elevation he can determine by means of stereoscopic photographs and if this is 100 microns or less. The clinical observer has to theorize concerning the third dimension. That is one of the chief reasons why there is a variation of opinion in regard to the exact nature of some of these fundus diseases. It forces one to talk in the terms of streaks, spots, white areas, and clinical diagnoses instead of in terms of fibrosis, gliosis, migration of pigment, exudate, and transudate. We can establish the true nature of the process by obtaining the third dimension of the lesion — that is by study of microscopic sections, which alone gives the third dimension in any degree of accuracy.

If the fourth dimension is time, then this also may be obtained by comparing the details of photographs taken at different intervals of time.

Dr. Bedell correlated the fundus picture and the visual fields in cases of blocked branch of the central retinal artery before the American Ophthalmological Society last June. A correlation between fundus pictures and findings in microscopic picture depends on the availability of material for such microscopic study. This correlation can be made more possible by pathological study of all enucleated eyes even though in every instance the fundus has not been studied. It was by routine study of such eyes that the true nature of retinitis pigmentosa, circinate retinitis, and other conditions has been determined only fairly recently.

The following correlations of fundus picture and microscopic findings illustrate only crudely what can be done (slides were shown):

1. In retinitis pigmentosa the pigment epithelium of the retina is profoundly diseased and even absent in places. Pigment in the retina has migrated from the pigment epithelium. It is found in places surrounding some of the vessels and their branches producing the typical bone corpuscle form. The choroidal vessels are not sclerosed. The loss of vision is due to destruction of rods and, later, cones. The attenuated vessels are due to thickening of the adventitia.

2. Black spot in macula in myopia is due to proliferation and migration of pigment epithelium of retina.

3. Pigmented streaks in fundus following chronic separation of choroid are due to proliferation of pigment epithelium of the retina while the choroid is off due to folds in pigment epithelium.

4. Angioid streaks are due to fibrosis and contraction of outer two-thirds of the choroid, throwing the chorio-capillaris and pigment epithelium into folds. This pathological change cannot be an artefact. It will produce the ophthalmoscopic picture of angioid streaks as well as clinical symptoms. It is a rare and unique microscopic picture. There is not another one of these in the ten thousand specimens at the Pathological Laboratory of the Massachusetts Eye and Ear Infirmary. It is not easy to understand why this first tangible evidence of pathological changes is not generally accepted. The objections are on such weak grounds as — it was not seen with an ophthalmoscope before the eye was enucleated, the fellow eye had no typical streaks, and the pathological findings do not happen to agree with the theories based on two dimensional study. If you say this is not the pathological picture of angioid streaks, it is even a more rare condition than angioid streaks. I am likewise completely convinced that angioid streaks are associated with osteitis deformans as well as with pseudoxanthoma elasticum. Osteitis deformans can be ruled out only by X-ray study of every bone in the body including the small bones of the hands and feet and by the determination of the amount of serum phosphatase. Unfortunately, few laboratories are prepared to determine the serum phosphatase value.

In closing, may I quote from the essayist in one of his previous papers? "The correlation of the photographs and the pathological condition are essential to the solution of the problem." But I should like to pleuralize the last word of the quotation.

DR. ARTHUR J. BEDELL, closing: I feel that I have been taken back many centuries to the philosophical stage of the development of medicine. Dr. Terry says that it is up to us to prove that he is in error. I believe you will agree with me that the burden of proof lies upon him. He showed a drawing of retinitis pigmentosa. Why he did so after having seen the beautiful photograph is beyond my comprehension, for his drawing was schematic, and, had the fundus been photographed, I am convinced that not even the distribution of the large vessels would have corresponded to those in the sketch. In answer to his question as to how small an elevation I can measure stereoscopically, I regret that I am unable to answer, for most of my patients keep their eyeballs.

That the third dimension is demonstrable in the stereoscopic fundus photographs is verified by all who have taken the time to study them. When photographs are taken of all fundus lesions and compared with the laboratory findings, a long step will be taken toward the solution of many of our present problems in fundus interpretation.

I thank Dr. Fry for his interest and discussion, which has added much to the value of the presentation, and I deeply appreciate Dr. Terry's exposition which is evidence of his thorough investigations.

DEFECTIVE CENTRAL VISION FOLLOWING SUCCESSFUL OPERATIONS FOR DETACHMENT OF THE RETINA*

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NEW YORK

THIS PAPER presents the result of an effort to determine the cause of the poor central vision so frequently obtained after successful operations for detachment of the retina. The explanation seems to evolve around the tendency for a detached retina to undergo cystic degeneration, especially in the macular region, and it is this theme that will be discussed.

It is common experience that, following a successful operation for detachment of the retina, the central vision is frequently defective. The statistics of Dunnington and Macnie, compiled from the operated cases of retinal detachment at the Institute of Ophthalmology, New York City, were reviewed with the idea of determining what percentage of the reattached cases get normal central vision, and whether or not the duration of the detachment affected the central vision after reattachment. Of the fifty-five cases with reattachments there were eighteen, or 32%, which obtained a central vision of 20/30 or better. There were thirty-two cases in which the duration of the detachment had been one month or less, and of these, twelve, or 38%, obtained a central vision of 20/30 or better. There were twenty-three cases in which the duration of the detachment had been over one month, and of these, six, or 26%, obtained a central vision of 20/30 or better.

From these figures then, one can deduce that approximately normal vision (20/30) is obtained in only about one-third of the successful cases,† and that the duration of the detachment is a factor in the visual result.

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* From the Institute of Ophthalmology of the Presbyterian Hospital, New York City.

† It would naturally be desirable to know the difference in the visual acuity obtained in those cases of reattachment in which the macula is detached, and those in which it is not. This does not seem practicable to obtain,

From the microscopic examination of the routine cases of retinal detachment which come to enucleation, it is well known that cystic spaces occur in the macular region, and, to a less extent, elsewhere in the retina. This change is usually considered a very late sequela, and its occurrence has been noted almost invariably in eyes in which there was other pathology, so that it is difficult to say to what extent detachment alone was responsible for the cysts, and to what extent other factors.

The detachments of the retina which usually come to microscopic examination are of two kinds:

1. The traumatic, the myopic, and the idiopathic types of very long duration in eyes which have developed an iridocyclitis and secondary glaucoma or atrophy of the globe; or those in eyes which have developed an iridocyclitis and secondary glaucoma or atrophy of the globe following repeated operations for detached retina.
2. The secondary types which occur as the result of contracture of fibrous tissue produced by the organization of an ectogenous or endogenous inflammatory exudate or hemorrhage, or as the result of a choroidal tumor or choroidal hemorrhage.

In most of these instances the retinal detachment has been present for such a long time when the eye comes to enucleation that advanced degenerative changes have taken place in the retina, and it is difficult to determine which of such changes are due to the retinal detachment alone and which are due to the other complicating features, such as atrophy of the globe, glaucoma, hemorrhage, inflammation and organization. Therefore, an effort has been made to study early cases of detachment of the retina in which there are no complicating features. The best opportunity to examine such cases is afforded by the instances when an eye with a simple serous detachment of the retina is enucleated because a sarcoma of the choroid is suspected. Such occasions are rare, and my studies are based upon twelve cases which have been gathered from various sources. Five are from the Registry of Ophthalmic Pathology at the Army Medical Museum, and the

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however, because sometimes the protrusion of the detached retina prevents a view of the macula, and often there is a very flat, slit-like detachment difficult to determine, especially if the vitreous is cloudy. Anyway, our records do not make specific mention of whether or not the macula is involved unless some pathology is noted.

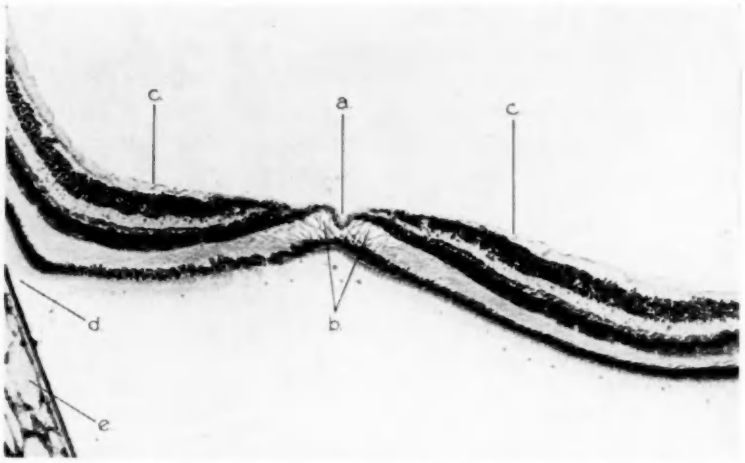

FIG. 1 — (Reese) — Case 1 (see chart). The greatest detachment of the retina is below and it extends temporally including the macular region. Elsewhere in the eye there is a flat detachment. (a) Fovea centralis. (b) Cystic spaces in the external plexiform layer (Henle's fiber layer) radiating around the fovea centralis. (c) Folds or wrinkles in the internal limiting layer. (d) Limit of detachment in macular region. (e) Choroid.

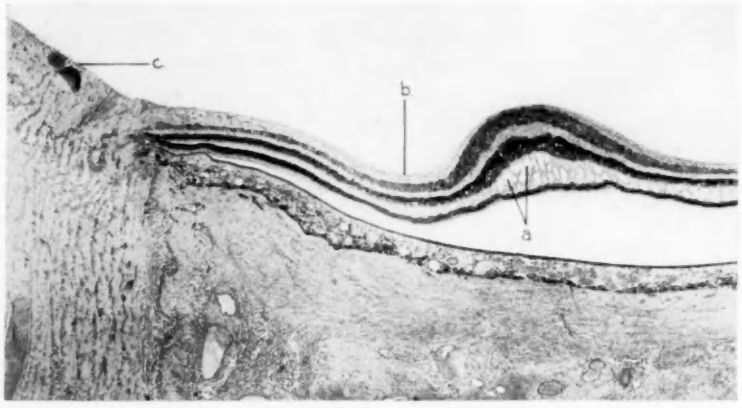

FIG. 2 — (Reese) — Case 2 (see chart). There is a flat detachment of the retina over the temporal side. The section is through the macular region but not through the fovea. The entire temporal portion of the retina is thickened by edema which separates the various retinal elements. (a) Cystic spaces in the external plexiform layer. A few small cystic spaces can be seen in the internal nuclear layer. (b) Folds and slight detachment of the internal limiting layer. (c) Disc.

others are from the Wills Eye Hospital, Wilmer Eye Institute, Massachusetts Eye and Ear Infirmary, and the Institute of Ophthalmology, New York City.

These twelve cases of simple serous detachment of the retina, ranging in duration from less than two weeks to four years, all showed cysts in the macula, Figs. 1, 2, 3, and 4. It is here that they seem to appear first and to attain their largest size. In the older cases, of three months or longer, they are frequently also found elsewhere in the retina. Their location is primarily in the external plexiform layer but they are often seen to a lesser degree in the internal nuclear layer. *At first, the retinal elements seem to be merely separated by edema which localizes into small cystic spaces.* These coalesce so that ultimately the cysts become larger and separated from contiguous cysts by thin septa of glial tissue. The content of the cysts is usually a homogeneous, colorless fluid which stains weakly with eosin in some instances, particularly when the condition is of long standing. This no doubt represents an increase in the albuminous content due to inspissation.

The cysts present in the retinas of all twelve cases studied seem to be secondary to the detachment. There is no inflammatory element present. There is a history of trauma in only three of the cases, and one of these is in the nature of a blow on the top of the head three months prior to the onset of the detachment. Glaucoma was present in four of the cases. An operation for reattachment of the retina was performed in two cases but the changes incident to this did not seem to be a factor in the formation of the cysts.

The edema of the retina and the cyst formations are probably due to the altered retinal circulation which permits a stasis of tissue fluids. This is more prone to appear in the macula first and more severely because this area is relatively avascular. When a detached retina becomes reattached, the gradual disappearance of the edema probably accounts for the slow progressive improvement in central vision and in the peripheral fields which may take place over a period of months. If cyst formations in the macula have led to a destruction of the neurones, then a permanent defect in central vision is to be expected.

The presence of cysts in the macula or elsewhere in the retina cannot be recognized clinically as such by the ordinary ophthalmoscopic examination. When present, they may appear as poorly demarcated, grayish to yellowish-white foci, or as a rarefied area described usually as a "hole." Vogt has recognized them as cystic

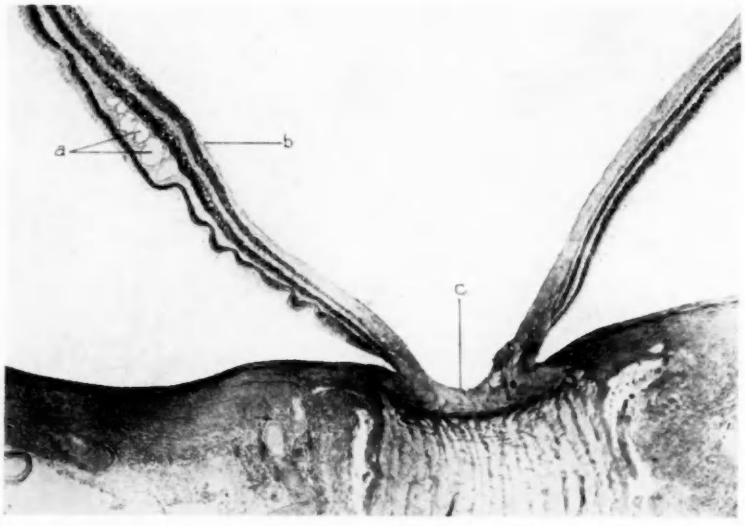

FIG. 3 — (Reese) — Case 4 (see chart). There is a complete detachment of the retina. The highest site is below, and above there is a flat detachment. (a) Cystic spaces in the external plexiform layer with a few very small daughter cysts in the internal nuclear layer. (b) Folds in the internal limiting membrane. (c) Disc.

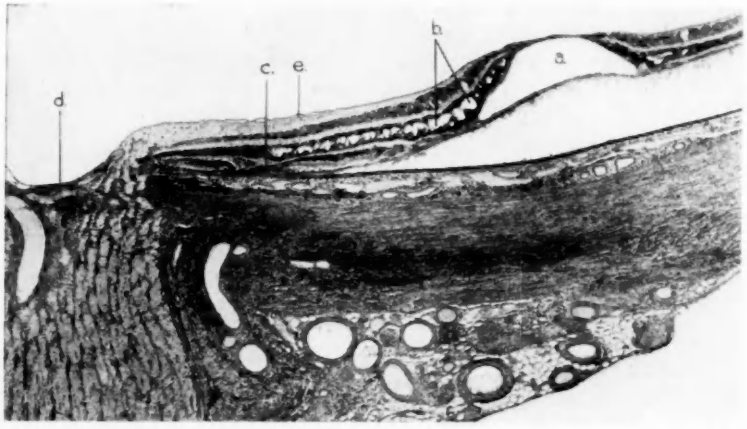

FIG. 4 — (Reese) — Case 12 (see chart). The entire retina is detached. The highest area is on the nasal side and a more or less flat detachment is present temporally including the macular region. (a) A large cyst exactly at the fovea centralis. The anterior and posterior walls are formed by a thin layer of glial tissue. The rods and cones are missing over the site corresponding to the cyst. (b) Daughter cysts in the internal nuclear layer. (c) A site between the macula and the disc where the rods and cones are missing. They are replaced by a layer of glial tissue which is adherent to the underlying pigment epithelium. This is the limit of the detachment and these are reparative changes occurring as a result of friction between the retina and choroid. (d) Physiological cup of optic disc. (e) Folds and slight detachment of internal limiting layer.

spaces by means of the red-free light, and has described their appearance in cases of retinitis pigmentosa, thrombosis of the central vein, and detachment of the retina.

In cases of detachment of the retina, I find that a cystic degeneration of the macula can be recognized if this area is sufficiently close to the underlying choroid to permit a reflection of a sharp narrowed beam of light from the choroidal surface. This beam of light, which is afforded by the usual ophthalmoscope, is directed to one side of the macular region to be observed, and the observer's head shifted until the incident rays reflected from the choroid are detected in the non-illuminated field. This is comparable to retro-illumination of the cornea by reflected light from the iris in slit-lamp examination. When this method is used in suitable cases, that is, detachments of the macula which are sufficiently close to the underlying choroid to permit a reflection of light from the choroidal surface, quite frequently one can detect rarefied areas of the macula, the borders of which are out-

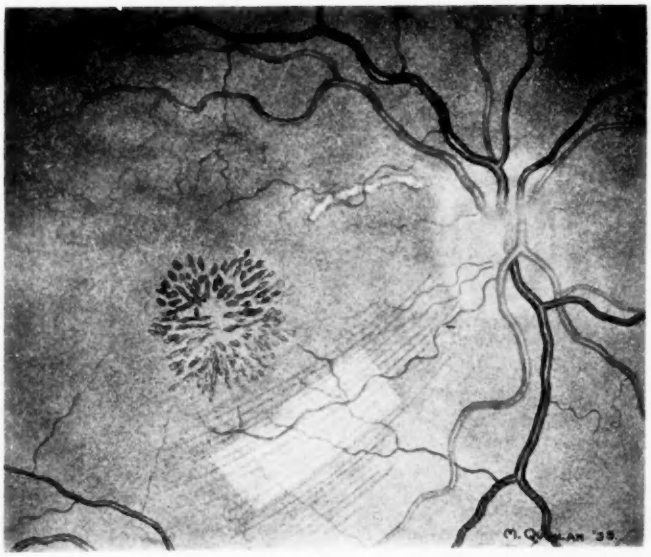

FIG. 5 — (Reese) — A drawing of the macula seen by retro-illumination with the ophthalmoscope in a case of detachment of the retina the duration of which was six weeks. The involved area was mostly over the lower portion of the fundus but a flat detachment extended over the macular region. Patient, aged 26, gave no history of injury, and no hole was seen. The rectangular beam of light is seen below, and in the macula are linear shadows radiating around the fovea centralis. These represent cystic spaces in the external plexiform layer. There is a white line coursing horizontally between the upper border of the disc and the macular region which probably was the limit of the detachment and represents glial repair occasioned by the friction of the rods and cones over the choroid (Fig. 4, c).

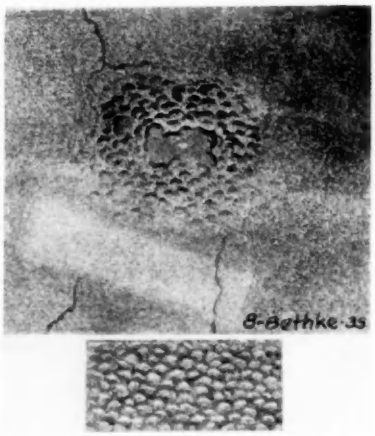

FIG. 6 — (Reese) — A drawing of the macula seen by retro-illumination with the ophthalmoscope in a case of detachment of the retina. The duration was undetermined as the patient was six years old. No history of trauma. A hole, around which there were some low grade chorio-retinitic changes, was noted in the periphery of the fundus below. The rectangular beam of light is seen below and in the macula there is a rarefied central area or so-called hole with irregular borders and surrounded by cystic spaces. In the center of the rarefied area at the site of the fovea centralis is a small light reflex presumably from the anterior limiting layer of a central cyst (Fig. 4, a). The small insert below is the Scotch-grain leather appearance of the retinal surface seen in this case, particularly on the nasal side. This frequent appearance of a detached retina is due to the wrinkling of the internal limiting membrane.
(Figs. 1, c; 2, b; 3, b; and 4, e).

lined by dark shadows, and radiating in most instances around the fovea, Fig. 5. These must represent cystic spaces in the external plexiform layer (Henle's fiber layer) of the retina because this is the layer in which the cysts were found in the microscopic sections and this is the only layer of the retina the structure of which radiates from the fovea. In more advanced cases there may be an irregularly round rarefied area at the fovea surrounded by cystic spaces and apparently formed by their confluence, Figs. 4 and 6. Such so-called "holes" can, of course, be seen by the ordinary ophthalmoscopic examination, but the cystic element around their periphery can be made out best by the method of retro-illumination. The lesions described here have also been studied by means of the red-free light and the Friedenwald slit-ophthalmoscope, but these methods in my hands do not give any additional information. When a macula which has a cystic degeneration becomes reattached following an operation, I have not been able to recognize the cysts as such, but they usually appear as poorly demarcated, hazy, grayish to yellowish-white foci. In some cases, after a few months, pigmentary changes appear in the macula.

In most instances, what is diagnosed clinically as a hole in the macula certainly represents conglomerate or confluent cysts.* This is based on the following facts:

1. One often sees in the center of such so-called holes a foveal reflex which apparently comes from the anterior limiting layer of the cyst, Fig. 6.
2. By means of red-free light, or by retro-illumination with the ophthalmoscope, one can usually see cystic spaces surrounding the so-called hole, Figs. 4 and 6, and sometimes it can be detected that the irregular borders of the hole are continuous with the surrounding cysts. Occasionally, septa can be seen in the "hole."
3. Actual holes in the macula almost never are seen in microscopic sections whereas cystic spaces are very common.

Conclusions

1. A cystic degeneration of the macula accounts for the poor central vision after the reattachment of a detached retina which involved the macular region.
2. The defective central vision so frequently found following successful operations for detachment of the retina suggests that a macular detachment with cyst formation was present even though it might not have been recognized clinically.

* * *

I wish to thank Doctor John M. Wheeler for his kindly interest. Technical work and microphotography by Mr. N. E. Ross. Drawings by Mr. Gustav Bethke and Miss Marjorie Quinlan.

* Cysts and/or holes may also appear in the macula in the following conditions:

1. Following trauma. This is thought by most to be due to a localized edema which leads to a pressure atrophy of the affected retinal elements, forming small cystic spaces the walls of which coalesce to form larger ones. Sometimes the cyst spaces are filled with hemorrhage. A hole may be present after a commotio-retinæ disappears.
2. As a complication of an iridocyclitis. Here toxins apparently reach the macular region and give rise to an inflammatory edema. These cysts may be transitory and even reappear.
3. Accompanying retinitis pigmentosa. There may be cystic formations in the macula, usually in the early stage of the disease. These may be transitory and are usually attributed to edema.
4. Following occlusion of the central vein of the retina. Cysts in the macula are extremely common in this disease. They are due to edema and sometimes they are filled with blood.
5. As a complication of retinal arteriosclerosis. These occur in older people and are attributed to edema on an arteriosclerotic basis. They may appear in the early stage of a central senile macular degeneration. Arteriosclerosis on a syphilitic basis may also be the cause of macular cysts.

The Results Relative to the
Occurrence of Retinal Cysts in Twelve Eyes Enucleated with Simple Serous Detachment of the Retina

CASE NO.	DURATION OF DETACHED RETINA	DETACHMENT OF MACULAR REGION	CYST PRESENT IN MACULA	CYST PRESENT ELSEWHERE ALSO	CAUSE OF ENUCLEATION	HISTORY OF TRAUMA	GLAUCOMA PRESENT	OPERATION FOR DETACHED RETINA
1 (See Fig. 1)	2 weeks or less	Yes	Yes	No	Choroidal sarcoma suspected.	Yes. Hit on top of head 3 mos. prior to detachment.	No	No
2 (See Fig. 2)	2 weeks	Yes	Yes	No	Choroidal sarcoma suspected.	No	No	No
3	1 month	Yes	Yes	No	Choroidal sarcoma suspected.	No	No	No
4 (See Fig. 3)	2 months	Yes	Yes	No	Choroidal sarcoma suspected.	No	No	No
5	3 months	Yes	Yes	Yes	Choroidal sarcoma suspected.	No	No	No
6	6 months	Yes	Yes	Yes	Choroidal sarcoma suspected; secondary glaucoma causing pain.	No	Yes	Yes

CASE NO.	DURATION OF DETACHED RETINA	DETACHMENT OF MACULAR REGION	CYST PRESENT IN MACULA	CYST PRESENT ELSEWHERE ALSO	CAUSE OF ENUCLEATION	HISTORY OF TRAUMA	GLAUCOMA PRESENT	OPERATION FOR DETACHED RETINA
7	9 months	Yes	Yes	Yes	Choroidal sarcoma suspected.	No	No	No
8	9 months	Yes	Section not exactly through macula but cysts present in retina temporal to disc.	Yes	Choroidal sarcoma suspected; secondary glaucoma causing pain.	No	Yes	No
9	1-2 years	Yes	Yes	Yes	Choroidal sarcoma suspected; secondary glaucoma causing pain.	Yes	Yes	No
10	2 years	Yes	Yes	Yes	Choroidal sarcoma suspected; secondary glaucoma causing pain.	No	Yes	No
11	Several years	Yes	Yes	Yes	Choroidal sarcoma suspected.	Yes	No	Yes
12 (See Fig. 4)	4 years	Yes	Yes	No	Choroidal sarcoma suspected.	No	No	No

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DISCUSSION

DR. CLIFFORD B. WALKER, Los Angeles: I will try to be very brief in discussing this most excellent paper which elucidates still further for our use some information and comes at a very timely interval when we should be studying the macula in connection with separated retina and also, perhaps, in connection with tumors because the conditions there offer an excellent indication of what is going on.

The cystic degeneration that occurs in Henle's layer, that is, the plexiform or internuclear layer, was first noted by Bruno in 1902 in connection with melanosarcomata of choroid. That was repeatedly noticed until 1910, and then in 1916 Vogt noticed the cystic degeneration taking place at the macula in the case of carcinomata and wished to connect it with separated retina and the possibility of showing the mechanism of the detachment.

Dr. Reese did not have time to mention some of the facts that might be noted in this connection. I would like, for instance, to exhibit the last slide that was shown and have you notice a further possibility; namely, the laminating or layering possibility of hole formation. You can see that the layer of Henle, which is quite marked at the macula and constitutes most of the tissue there, because the two layers have been pushed aside, has gone in such a way that you have a top layer of limiting membrane and a bottom layer and there are intermediate stages, as you can well imagine, which might occur with the external limiting membrane in such a way that three layers may occur, laminated holes, we will say for lack of a better phrase right now.

I wish to discuss the mechanism of that process a little further, although it is practically obvious from a slide like that, that the top layer might be pulled off and a hole formed there and a light reflex might be possible from the bottom.

Again, the reverse might occur in a thin place which I noticed on the outer angle. At some time a hole might appear there and yet the top not be perforated.

In order to show you how this may be examined, I will show you an instrument in a moment. But first, I would like to speak on this other slide.

We will speak this time concerning the pebbling that you notice around the hole. If this is cystic in character it is exceedingly regular in outline. But the cysts, as you notice them, are not quite so regular, not quite so uniform, not quite so able to indicate, as in the lower drawing, the pebbling like Scotch grain leather.

So, we might seek further information on that point. As you look at this hole in the center you seem to detect a second layer present. The absence of the first layer is something to consider. Still, there might not be sufficient leakage there to give a high elevation and a second layer able to give a light reflection. Those possibilities need to be considered because they actually occur.

In order to see them as well as possible, I will show a little device that I have attached to the strongest ophthalmoscope now available, the Friedenwald. (Slides were shown).

The pebbling is due to toxic effects. When one watches the same thing in capillary degeneration there is edema, with early stages of cystoid degeneration. But, with that occurring under the conditions found in the retina, it seems to me a thing well to remember in connection with the pebbled effect that is noticed. That is to say, it seems reasonable to regard that pebbling largely as a pattern of the basework of the retina and as an underlying edema and pyoid degeneration rather than cysts in that particular spot and that particular arrangement.

Cystic degeneration chooses the internuclear layer first; second, it chooses the nuclear layer; and third, it chooses the external nuclear layer. It is a nutritional phenomenon.

DR. HARRY S. GRADLE, Chicago: The lack of central visual acuity following a successful detachment operation may be due to one of three things. In the first place, there may have been a preoperative disease involving the macula. This possibly we might have seen beforehand or if the detachment was sufficiently extensive to elevate the macula it is impossible to observe this.

Second, a postoperative change in the macula is usually subsequent to hemorrhage. Hemorrhage into the macula following detachment operation is not uncommon.

Thirdly, cases that under ordinary ophthalmoscopic examination show perfectly normal maculæ still have a lack of central visual acuity and a very definite central scotoma often three to five degrees in size.

Since April, I have seen four of these cases in which I have been able to detect the cause of the failure of central vision exactly as has Dr. Reese; namely, as cyst in the macula. I have not been able to see these with the ordinary ophthalmoscope, but with the Friedenwald slit-ophthalmoscope, when used as Dr. Reese has described, by retro-illumination, if you wish to call it that, and particularly when used with the red filter — and I said red emphatically, not red-free. The details of such cysts do not appear in the red-free filter, but appear clearly when the red filter is used.

In these four cases the cysts lay directly in the macula and varied from one-quarter to a little more than one-third disc diameter in size. In every instance they were regular and oval, vertically oval. This was probably a coincidence. At no time do I find the irregular walls depicted as in the last picture of Dr. Reese's. They were elevated and that elevation could be detected by the use of the extremely fine slit. There seemed to be a rather thin but very definite anterior wall which, apparently, varied in thickness in the different cases. The surrounding macula was flat and

as far as could be detected there was no elevation of the retina in or around the macula except at the cyst.

In the other case which has now been under observation for five months, there is beginning to develop a very thin line of pigment dots along the upper inner border of the cyst. Ordinarily, you know, there is a definite pigment flow, if we may call it that way, from the periphery of the retina toward the optic nerve, but in this instance the pigment is appearing at the inner upper margin of the cyst.

One of the cysts lies slightly excentric to the macula and borders on the lower outer border of the macula. These cysts explain fully the lack of central vision, explain fully the central scotoma that exists. Interestingly enough in all of these cases the vision could be brought to a useful point by the use of telescopic lenses, although, of course, they were not ordered in these cases.

I think that Dr. Reese has filled a very important niche in our knowledge of the lack of central visual acuity following otherwise successful operations for detachment of the retina.

In this connection may I speak of one other observation that I have seen in two cases; namely, subsequent to detachment, or operation for detachment of the retina, on the fifth to ninth day, there has appeared a massive detachment of the choroid. This has persisted in one instance for four weeks. In another instance now for three weeks, and was followed by a very successful reattachment of the entire retina with good central visual acuity.

From the mechanical standpoint it is very easy to see why a choroidal detachment is a desirable feature, because, after all, whatever surgery we perform for detachment of the retina is for the purpose of producing an adhesion between the retina and the choroid for the production of that adhesion for which the retina must either be in opposition with the choroid or very close thereunder.

If a large amount of subretinal fluid is present, it is obviously impossible for the retina to be caught in the adhesive process and retained there. Consequently, there is the possibility that certain types of retinal detachment may be handled by the artificial production of a detachment of the choroid, which, of course, is spontaneously limited.

RECENT ADVANCES IN THE SURGERY OF CHRONIC GLAUCOMA

OTTO BARKAN, M.D.

SAN FRANCISCO

INTRODUCTION. A preliminary report of a new surgical procedure for glaucoma, consisting of operating on Schlemm's canal under direct magnified vision, was presented before the Section on Ophthalmology of the American Medical Association[1] and read before the Association for Research in Ophthalmology at Kansas City in May, 1936.[2] In the present article the subject matter is approached from another point of view and certain observations and deductions on the pathologic-anatomic entity of chronic simple glaucoma are added in the hope that they may be verified by other observers.

FACTORS OF SUCCESS IN SURGERY

Success in surgery is dependent largely on two factors: knowledge of the cause of the disease and the direct visibility of the area under operation. It has been the constant aim of surgeons to gain visual access to the hidden recesses of the human body. These efforts have been crowned with success in various regions through the invention of instruments which by means of mirrors, prisms, and improved sources of light deflect the incoming and outgoing rays in such a way that the surgeon is afforded direct vision of a field of operation which was formerly hidden from view. Largely because of these methods of operating under direct vision, the successful surgery of the larynx, bronchi, urinary bladder, prostate gland and other regions has been made possible.

OBSTACLES TO THE SUCCESSFUL SURGERY OF CHRONIC GLAUCOMA

The surgery of glaucoma has not kept pace with these advances. Ophthalmologists have continued to labor under two great handicaps: firstly, the lack of knowledge of the cause of the increased intra-ocular pressure, and, secondly, the necessity of operating

blindly or semi-blindly, for, after the incision of the external tunic of the eye is made in a case of glaucoma, the further course of the operation is hidden from view by the opaque scleral limbus. In glaucoma the surgeon cannot see and he cannot more than guess what effect his instruments will have inside the eyeball nor does he know the cause of the condition he is trying to relieve.

RESULTS OF RECENT INVESTIGATIONS

These handicaps, it would appear, have in large measure been overcome in a majority of cases of chronic glaucoma[2] (chronic glaucoma Type I of the writer or "chronic simple glaucoma") by:

(1) *Establishment of the mechanical cause of the increased intra-ocular pressure.* Slit-lamp bio-microscopy of the angle of the anterior chamber shows the pores of the corneo-scleral trabeculum to be blocked in this type of case.[3] Removal of the blocked trabeculum by the author's procedure of opening Schlemm's canal under direct vision reduces the increased intra-ocular pressure to normal. The effect of the operation indicates that the increased intra-ocular pressure is the direct result of stoppage of outflow through the pores of the trabeculum. This trabecular block has been found to be present in all cases of chronic glaucoma Type I (chronic simple glaucoma) examined bio-microscopically to date.

(2) *Operation under direct vision and removal of the cause of the increased intra-ocular pressure.* The blocked trabeculum is incised from within the eyeball, the trabeculum being kept under full view during the operation by means of a contact glass devised by the author for this purpose. The physiological direction of outflow of aqueous from the anterior chamber into Schlemm's canal is thus restored.

A similar approach to the angle across the anterior chamber was used by the Italian, De Vincentiis, in 1891.[4] He, however, carried out his procedure unable to see where his knife was going. the operation was later abandoned.[2]

BIO-MICROSCOPY OF THE ANGLE OF THE ANTERIOR CHAMBER

The technique of slit-lamp bio-microscopy of the angle which was devised for these investigations and which was used to establish the preoperative diagnosis has been published in a previous article.[3] Since then the ceiling model there described has been im-

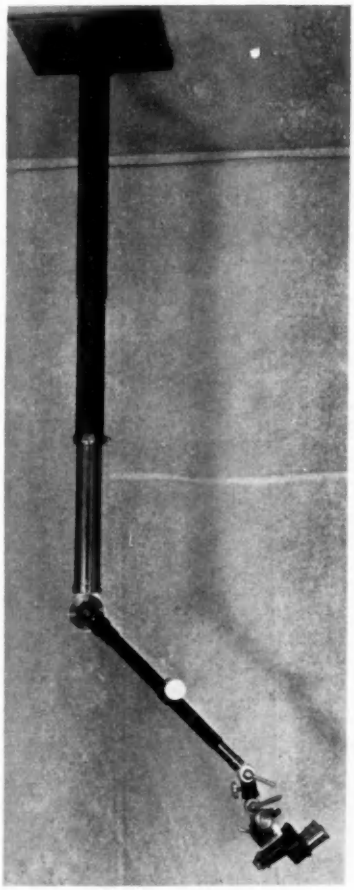

FIG. 1 — (Otto Barkan) — Binocular micro-gonioscope — ceiling suspension model.

FIG. 2 — (Otto Barkan) — Binocular micro-gonioscope — floor model.

proved by the adoption of various mechanical devices so as to make it even more flexible while maintaining its steadiness and decreasing its weight (Fig. 1). In addition to this model which has the advantage of not occupying floor space, a simplified unit has been devised for use on a moveable floor stand (Fig. 2). This floor unit is equally flexible and possesses the advantage that it may also be used as a corneal microscope for routine examination of the anterior segment while the patient sits upright or reclines in a treatment chair with head rest. Both units may be fitted with

the head of either a Zeiss or a Bausch and Lomb corneal microscope. The use of 2 × objectives and 10 to 12½ × oculars when combined with the refractive power of the eye and the contact lens afford a total magnification of up to nearly 40 ×. This technique, in the opinion of the writer, will be found superior to and much simpler than any of the previous gonioscopic methods used to examine the angle of the anterior chamber.

As the blocked trabeculum (the trabecular pigment band of the writer) (Fig. 3) and the thickening or sclerosis of the trabeculum have not been described before with the gonioscopic methods of the past, they would seem to be inadequate. It seems necessary, therefore, for the satisfactory investigation of these structures to use a technique which will show, as does the one mentioned above, details of the trabeculum and of the trabecular pigment band.

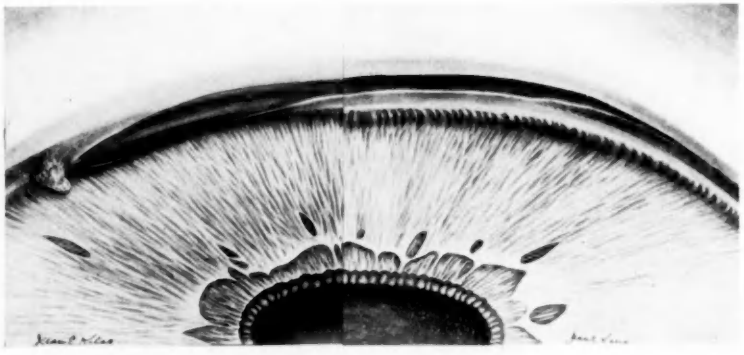

FIG. 3 — (Otto Barkan) — Drawing representing incision of the trabeculum or opening of Schlemm's canal as it appears upon examination with the writer's micro-gonioscope. The trabecular pigment band is shown at the beginning and the end of the incision. The small lump at the left consists of coiled up remnants of the trabeculum.

MAGNIFICATION

For purposes of quick inspection and orientation, the angle may be conveniently viewed through the contact lens with an ordinary binocular 5 diopter head loupe. This affords 2 × to 3 × magnification. For greater detail, however, the bio-microscopic technique described above should in my opinion be used as routine, just as the corneal microscope is routinely used for examination of the anterior segment.

ILLUMINATION

The angle of the anterior chamber may be advantageously illuminated in two ways:

(1) By direct illumination in which the incident beam comes from more or less the same direction as the line of regard of the observer.

(2) By indirect or transscleral illumination in which the beam comes from the opposite direction and traverses the sclera on its way to the angle, thus transilluminating it ("posterior gonioscopy" of Trantas).

In using direct illumination and for purposes of orientation and observation of a relatively large field I have devised a hand lamp with a beam 5 to 10 mm. in diameter to answer the purpose. Detail is enhanced by cutting down the beam of light to a slit. For actual bio-microscopy I use a Vogt carbon arc slit-lamp mounted on an adjustable stand. Transillumination is performed with the narrow beam of my hand lamp, cut down to a spot 3 mm. in diameter. I am at present developing improved devices for the illumination of the angle of the anterior chamber which will be published in the near future. Castroviejo[5] has recently described a light attached to the head of the microscope which he has used for the purpose of photographing the angle and examining it, the microscope being held in the hand.

CHRONIC SIMPLE GLAUCOMA AS A PATHOLOGIC-ANATOMIC ENTITY

The writer believes that the group of cases in which this operation "under direct vision" has proved to be especially indicated and effective and which he has classified as chronic non-congestive glaucoma, Type I, constitutes a sharply circumscribed pathologic-anatomic entity. This entity appears to cover the hitherto somewhat ill-defined clinical picture of "chronic simple glaucoma." It furthermore appears to correspond to the clinical conception of "anterior glaucoma." This would seem to indicate that the latter is identical with "chronic simple glaucoma." In all cases of this group examined bio-microscopically to date, a block of the trabeculum appears to be the mechanical cause of the increased intraocular pressure.

As a result of these investigations which included, in addition to the usual examinations, (1) measurement of the depth of the anterior chamber with Ulbrich's drum, (2) bio-microscopy of the angle of the anterior chamber with the author's technique, and (3) observation of the effect of reestablishing the communication between the anterior chamber and Schlemm's canal by operation under direct vision, it would seem that a definite pathologic entity has crystallized, which in its early and uncomplicated stage and even in the last stage of blindness and absolute glaucoma presents the following characteristics:

(1) The depth of the anterior chamber is normal or only slightly reduced.

(2) The angle is open.

(3) The pores of the trabeculum are blocked. This block is apparent usually as an infiltration, with pigment granules, that forms what the writer has termed the trabecular pigment band. When, as is sometimes the case, little or practically no pigment is present, a block of the pores may still be inferred in these cases from the definite thickening of the trabeculum as shown by its lack of transparency to the beam of the slit-lamp and from the pressure reducing effect of opening the canal by incising the trabeculum. The cause of this sclerosis or impermeability of the trabeculum and of the block of its pores with pigment has not been determined. One wonders what the physico-chemical constitution of the aqueous may be, what hormonic, dietary, infectious or autonomic factors may be at work, or what effects are produced by disease of the end arteries which supply the region of the canal of Schlemm.[6] It is interesting to note in passing that a theory of pigment glaucoma was, in the past, suggested on the basis of isolated observations of infiltration of the angle with pigment as observed in anatomic sections by Levinsohn,[7] inter al., and of dissemination of pigment on the inner lining of the anterior segment as observed by Koeppe[8] with the corneal microscope and slit-lamp. Studies of anatomic sections of the cribiform ligament (corneo-scleral-trabeculum) led Henderson[9] to suggest a sclerosis or fibrosis of it as a primary cause of glaucoma.

(4) There is occasional atrophy of the pigment epithelium of the iris, or more rarely exfoliation of the lens capsule (Vogt).[10]

(5) Glaucomatous excavation of the optic nerve is present.

TRABECULAR PATHOLOGY AS THE CAUSE OF INCREASED INTRA-OCULAR PRESSURE

Further evidence that a block of the trabeculum is the primary mechanical cause of the increased intra-ocular pressure in chronic simple glaucoma and not a result of it is shown by those cases in which the pigment block is present not only in the affected eye but is also found in the healthy fellow eye which shows not the slightest other sign or symptom of early glaucoma and responds normally to all other clinical tests, including provocative tests and diurnal tension curves. It is worthy of particular note that *trabecular pathology is present in these otherwise apparently healthy fellow eyes before increased tension becomes evident.* The reason for this would seem to be that the stoppage of the pores has not yet advanced to the point of blocking outflow sufficiently to cause retention of intra-ocular fluid or to cause reflexly excess formation of fluid through an increased vascular response. The latter when it does occur may be interpreted, it would seem to me, as an attempt to overcome the block and maintain normal metabolic exchange.[11]

EARLY DIAGNOSIS

It is evident that the bio-microscopic method of examining the angle often enables a much earlier diagnosis of chronic simple glaucoma than has hitherto been possible for, as stated above, certain signs of the disease, such as pathological change in the trabeculum, may be present long before there is any increase of pressure. The increased intra-ocular pressure would seem to follow in point of time and to be secondary to the mechanical result of the glaucomatous disease.

ON THE ÆTIOLOGY AND SYMPTOMATOLOGY OF CHRONIC SIMPLE GLAUCOMA

The cause of the sclerosis and of the stoppage of the pores of the trabeculum in this type of glaucoma (Type I) is, as stated above, as yet a matter of conjecture. It would seem at the present time to be a matter of greater practical importance to establish and confirm the mechanical cause of the increased intra-ocular pressure since a mechanical derangement can be relieved by surgical means, thereby preserving the function of the optic nerve.

Usually no subjective congestive symptoms such as haloes, mists, fogs, or pain are associated with the retention of intra-ocular

As a result of these investigations which included, in addition to the usual examinations, (1) measurement of the depth of the anterior chamber with Ulbrich's drum, (2) bio-microscopy of the angle of the anterior chamber with the author's technique, and (3) observation of the effect of reestablishing the communication between the anterior chamber and Schlemm's canal by operation under direct vision, it would seem that a definite pathologic entity has crystallized, which in its early and uncomplicated stage and even in the last stage of blindness and absolute glaucoma presents the following characteristics:

(1) The depth of the anterior chamber is normal or only slightly reduced.

(2) The angle is open.

(3) The pores of the trabeculum are blocked. This block is apparent usually as an infiltration, with pigment granules, that forms what the writer has termed the trabecular pigment band. When, as is sometimes the case, little or practically no pigment is present, a block of the pores may still be inferred in these cases from the definite thickening of the trabeculum as shown by its lack of transparency to the beam of the slit-lamp and from the pressure reducing effect of opening the canal by incising the trabeculum. The cause of this sclerosis or impermeability of the trabeculum and of the block of its pores with pigment has not been determined. One wonders what the physico-chemical constitution of the aqueous may be, what hormonic, dietary, infectious or autonomic factors may be at work, or what effects are produced by disease of the end arteries which supply the region of the canal of Schlemm.[6] It is interesting to note in passing that a theory of pigment glaucoma was, in the past, suggested on the basis of isolated observations of infiltration of the angle with pigment as observed in anatomic sections by Levinsohn,[7] inter al., and of dissemination of pigment on the inner lining of the anterior segment as observed by Koeppe[8] with the corneal microscope and slit-lamp. Studies of anatomic sections of the cribiform ligament (corneo-scleral-trabeculum) led Henderson[9] to suggest a sclerosis or fibrosis of it as a primary cause of glaucoma.

(4) There is occasional atrophy of the pigment epithelium of the iris, or more rarely exfoliation of the lens capsule (Vogt).[10]

(5) Glaucomatous excavation of the optic nerve is present.

TRABECULAR PATHOLOGY AS THE CAUSE OF INCREASED INTRA-OCULAR PRESSURE

Further evidence that a block of the trabeculum is the primary mechanical cause of the increased intra-ocular pressure in chronic simple glaucoma and not a result of it is shown by those cases in which the pigment block is present not only in the affected eye but is also found in the healthy fellow eye which shows not the slightest other sign or symptom of early glaucoma and responds normally to all other clinical tests, including provocative tests and diurnal tension curves. It is worthy of particular note that *trabecular pathology is present in these otherwise apparently healthy fellow eyes before increased tension becomes evident.* The reason for this would seem to be that the stoppage of the pores has not yet advanced to the point of blocking outflow sufficiently to cause retention of intra-ocular fluid or to cause reflexly excess formation of fluid through an increased vascular response. The latter when it does occur may be interpreted, it would seem to me, as an attempt to overcome the block and maintain normal metabolic exchange.[1]

EARLY DIAGNOSIS

It is evident that the bio-microscopic method of examining the angle often enables a much earlier diagnosis of chronic simple glaucoma than has hitherto been possible for, as stated above, certain signs of the disease, such as pathological change in the trabeculum, may be present long before there is any increase of pressure. The increased intra-ocular pressure would seem to follow in point of time and to be secondary to the mechanical result of the glaucomatous disease.

ON THE ÆTIOLOGY AND SYMPTOMATOLOGY OF CHRONIC SIMPLE GLAUCOMA

The cause of the sclerosis and of the stoppage of the pores of the trabeculum in this type of glaucoma (Type I) is, as stated above, as yet a matter of conjecture. It would seem at the present time to be a matter of greater practical importance to establish and confirm the mechanical cause of the increased intra-ocular pressure, since a mechanical derangement can be relieved by surgical means, thereby preserving the function of the optic nerve.

Usually no subjective congestive symptoms such as haloes, mists, fogs, or pain are associated with the retention of intra-ocular

fluid and increase of intra-ocular pressure in Type I. A reason for this would seem to be that the very gradual rate of formation of the block and the consequent very gradual onset and slow rate of increase of the intra-ocular pressure afford sufficient time for the ocular apparatus to compensate and adjust itself to the new order of things.

The meager precipitation of pigment from the clear aqueous and the correspondingly slow rate of its deposition to form the trabecular pigment band in chronic simple glaucoma may be regarded, it seems to me, as analogous to the gradual process of sedimentation occurring over the course of many years in a slow stream of very clear water. This conception illustrates the slowness of the development of the trabecular block and accounts for the slow rate of increase of the intra-ocular pressure over the course of many years, for the equally slow rate of formation of the excavation of the optic nerve, for the remarkable adjustment of the local vascular system and the development of a collateral circulation within the eyeball, and for the psychological adjustment of the individual himself to what may ultimately be an extremely high degree of mechanical block and of increased intra-ocular pressure. It would seem to account also for the practically asymptomatic course of the disease, for which reason it bears the name "simple glaucoma." It would seem probable then that this remarkable physical adjustment within the eye, with its concomitant absence of subjective symptoms, explains the well-known psychological adjustment of the individual as a result of which even an intelligent person not uncommonly loses the function of half or more of the optic nerve of one eye before he consults an oculist. Such is the insidious and dangerous nature of this disease. No alarm is sounded, in the shape of congestive symptoms, to warn the individual of the increase of intra-ocular pressure, of the progressing atrophy of the optic nerve and of impending blindness. In chronic glaucoma Type I the eye undergoes a slow process of pressure atrophy which is hardly ever associated with symptoms. When subjective symptoms and objective signs of congestion occur, which they may do rather exceptionally, they appear to be the result of an increase of the vascular response, and of an increased instability of the local circulation of the blood, or in other words of a change in the vasomotor factor rather than of a change in the rate of excretion or in the extent of the block of the outlet. This interpretation I believe holds true in Type I until its later stages, in which peripheral adhesions of

the iris have formed (Type III of the writer) and the eye is in the stage of venous decompensation. In these later stages of Type I, including "absolute glaucoma," the local intra-ocular arterial and venous blood pressures are increased sufficiently to withstand the increased pressure of the intra-ocular fluid upon them. As a result of this and through the development of a collateral circulation, the episcleral vessels dilate and a "caput medusæ" may form. Yet even in this late stage other congestive symptoms are not infrequently still lacking when the angle is open and the pathologic-anatomic picture is essentially the same as it was in the early stage except in degree.

OPERATIVE RESULTS

In chronic simple glaucoma (Type I of the writer) the increased intra-ocular pressure has been reduced to normal in every one of the twenty cases operated on to date in which post-operative bio-microscopic examination showed Schlemm's canal to have been opened over a sufficient extent of its circumference (about one-fourth). In a few cases in which the canal was missed the pressure remained unchanged. In others in which only a small part of the circumference of the canal was opened the pressure was only partially reduced. In those cases which were reoperated and the canal was struck, the tension was normalized.

NOMENCLATURE

For practical use and for the sake of brevity it would appear advisable to give a name to this procedure of opening Schlemm's canal under direct vision. I have on past occasions spoken of it as "goniotomy." A more apt and descriptive term for it would be, "trabeculotomy," I feel, for the operation consists precisely of an "incision of the trabeculum."

INDICATIONS

It is worthy of note that the reduction to normal of the increased intra-ocular pressure that occurs following this operation is irrespective of its height before operation. As the degree of increase of intra-ocular pressure does not appear to be in direct relation to the type of glaucoma, it should not in itself, in my opinion, be taken into consideration when choosing the type of operation for a given case. The pathologic-anatomic type should

rather be considered and the operation chosen according to it. Incidentally in this connection, and as a matter of fact, the operation of opening Schlemm's canal does happen to be particularly adapted to cases with high degree of increased intra-ocular pressure because only a puncture of the anterior chamber is made, no aqueous is lost and there is consequently no sudden reduction of pressure with its well-known dangers.

In the present state of our knowledge and judging by the writer's experience during the past year with this procedure, it would seem to be the operation of choice and worthy of trial in all cases of chronic glaucoma Type I with open angle (chronic simple glaucoma), irrespective of the degree of tension, whereas external filtering operations, iridectomy or cyclodialysis, would still seem to be indicated, at the time of this writing, in chronic glaucoma Type II[3] and in those cases which constitute the later stages of Type I and Type II in which organic peripheral adhesions of the iris have formed and signs of venous decompensation have supervened, Type III. However, those cases of Type III which constitute the later decompensated stage of chronic, non-congestive simple glaucoma (Type I) and which, therefore, began with a block of the pores of the trabeculum may perhaps be as successfully operated with this procedure, as is the early stage of Type I, by cutting through the peripheral adhesions of the iris at the same time as the trabeculum. In the future it may also prove feasible to operate with this technique on those cases of Type III that represent the late stage of chronic congestive glaucoma (Type II). The possibility of eroding or cutting through a major artery in a highly vascularized iris or of injury to a ciliary body that has been drawn forward behind the iris would, however, have to be borne in mind in this last mentioned Type III.

THE IMPORTANCE OF BIO-MICROSCOPY OF THE ANGLE FOR THE MANAGEMENT OF CHRONIC GLAUCOMA

The angle of the anterior chamber has, up to the present time, remained a much neglected region in spite of the important investigations of it that have been published over the course of many years by a small group of workers in this field. Among these may be mentioned in Europe, Trantas,[12] Salzmann,[13] Koeppe,[8] Thorburn,[14] Werner,[15] Vannas,[16] inter al., and in this country, Uribe Troncoso,[17] and more recently, Castroviejo[5] and Bruce.[18] Great credit is due Uribe Troncoso for his important investigations and

contributions in this field. To have acquired an intimate knowledge of the angle in the living has remained the distinction of this small group of investigators. It would seem that the inadequacies and difficulties of the "gonioscopic" methods of the past have prevented their more wide adoption and popularization. So, it happens that when speaking of glaucoma the ophthalmic surgeon of today, who has never seen and is unfamiliar with the region of the angle in the living, is more apt to visualize the external aspect of the limbus where he makes his incision rather than the inner aspect of the angle where the incision takes effect and where the functionally all-important structures are located. Interesting gonioscopic observations of trephined cases have been made by Troncoso and Reese.[19] Bio-microscopic examination of the effects of surgery on the topography of the angle as well as on its individual and neighboring structures, the iris, ciliary body, zonule, cornea and Descemet's membrane, is highly informative. *The importance of the visualization of the angle in the living by means of the binocular microscope (bio-microscopy) can, in my opinion, hardly be overemphasized.* With the new technique of bio-microscopy the examination of the angle is rendered easy and swift for both observer and subject. It should, I believe, be used routinely in indicated cases as the corneal microscope is used as routine for the examination of the anterior segment.

An intimate familiarity with the region of the angle of the anterior chamber will, I believe, be found to be of great value to every ophthalmic surgeon who operates within the angle either according to the blind or semi-blind methods of the past, or uses the new procedure of operating under direct vision.

SUMMARY

(1) The procedure of opening Schlemm's canal under direct vision involves a new principle in the surgery of glaucoma in that a hitherto hidden area of the inside of the eyeball (Schlemm's canal and the angle of the anterior chamber) is made visible during operation.

(2) The surgeon can see the area of blockage during the operation and can actually watch and guide his instrument in restoring the normal direction to the circulation of the intra-ocular fluid.

(3) The operation is without danger when the proper technique is used and has proved successful when certain preoperative indications (bio-microscopic diagnosis) have been fulfilled.

(4) The results are predictable and appear to be permanent. However, a much longer period of observation will be necessary to give a definite answer to the question of permanency of results.

(5) The operation is especially indicated in chronic simple glaucoma (Type I of the author). It is equally successful in certain cases of secondary glaucoma and promises to be applicable also in certain other cases of primary glaucoma.

(6) The pathologic anatomic entity described by the writer as chronic non-congestive glaucoma Type I appears to cover "chronic simple glaucoma" and "anterior glaucoma."

(7) Certain conclusions in regard to the ætiology and symptomatology of chronic simple glaucoma may be fairly drawn.

(8) Bio-microscopy of the angle of the anterior chamber is a useful adjunct to diagnosis and often enables an earlier diagnosis of glaucoma than has hitherto been possible. It would seem to constitute a considerable advance in the management of glaucoma.

(9) The operation of opening Schlemm's canal under direct vision may assist in the solution of the surgical problem of chronic glaucoma.

MOVING PICTURE DEMONSTRATION

This moving picture illustrates the operation described in my paper which consists of incising the corneo-scleral trabeculum from within the anterior chamber while it is in full view. In the three operations shown the surgical contact glass can be seen in position while the knife enters the temporal limbus, traverses the chamber and incises the trabeculum on the opposite side. The knife is then removed without loss of aqueous, the contact glass taken off and the eye padded.

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DISCUSSION

DR. JOHN M. WHEELER, New York: In the latter part of August I was privileged to be in Dr. Otto Barkan's office and to see a number of cases that he had operated upon. I found his scheme for examination very satisfactory, the arc-light for illumination and the magnification coming from the ceiling with an effective contact glass. I saw several iris angles. I saw the clefts, the caverns that he has described as a result of various types

of operations, and the incisions that he has made into the angle tissue according to the method that he has described. I also saw him operate, doing a goniotomy.

My own experience is limited to what I have done in the short period since the latter part of August. Dr. Barkan furnished me with his contact glass which enables the surgeon to enter the limbus without embarrassment from the glass. He also let me have one of the knives that he had made.

I operated first on cats' eyes and was assured that it was a perfectly safe thing for me to operate on the human eyes. I have operated on two of them. The first case was one that Dr. Barkan would consider at this stage unsuitable. It was a case of rather far advanced glaucoma with anterior synechia. It was not possible for me to see the drainage angle. The iris was crowded against the cornea, its peripheral part. However, I operated according to his method and had no trouble in separating the iris from the cornea, and whether I cut into Schlemm's canal or not I cannot say. Since the operation it is possible for one to see the little scar in the iris where the incision was made, the deeper anterior chamber. The tension went to low normal range and it has stayed there for a period of three weeks.

I operated three weeks ago today. There was very little reaction in spite of the fact that I got rid of anterior synechia, only a little hemorrhage which cleared up promptly, and, apparently, as near as one can judge at this stage, a successful result has been gotten.

There is an interesting thing that I think I might mention about operating on cats' eyes. I was bungling enough to destroy the point of the knife which Dr. Barkan had let me have. I used my discission knife and found it a perfectly satisfactory instrument for the purpose. So, I was interested a couple of days ago in reading the records on a dissertation on the subject to find that he said that cataract knives which had been ground many times until they were narrow and which had been ground so many times that the blade was short made satisfactory instruments for the operation in case one did not have the instrument that was designed for the purpose.

The second case that I operated on was one that Dr. Barkan would consider suitable, one in which the pigment band giving the location of the Schlemm's canal was easily seen. I made an incision which may or may not have been adequate. The tension has remained normal, just about average for a period of nine days. I operated nine days ago today.

That is not a very large experience in a short time, but I am convinced that the operation is not difficult. I had thought that it might be too difficult for most surgeons to attempt, but I became convinced before I operated on humans that it was perfectly possible for one to tell exactly where the point of the instrument is and for the surgeon to go exactly where he wishes with it. I was so convinced that I wondered if it might not be a good plan in making a section for cataract extraction to use a contact glass to obtain accuracy in location. It may not be necessary but it just occurred to me.

I am glad that Dr. Barkan spoke of the historical background of this operation and referred to the tradition that the operation has, because I think it would be a mistake for us to look upon it as a new operation. It certainly is not new. I think we might possibly, if we wish to be very

particular, question the type of paper today in which he speaks about physiological restoration of drainage of the aqueous, whether it is that or something else, I do not know.

The operation is a little older than Dr. Barkan remembers. I think I have read literature perhaps longer than he has. In 1891 Taylor reported a series of cases. That was Hugo Taylor, who was associated with De Vincentiis who suggested the operation. That was 45 years ago that he made a report on operations performed on dogs and on humans. He described the knife that was used, which must have been very much like the knife that Dr. Barkan has advocated. He described the cylindrical shank, a sickle-shaped blade, and even gave a formula for determining the proper size of the shank so that it would fit the opening made by the blade to exactly fill it without crowding the wound in the operation.

Before 1891, he operated on 12 dogs and reduced the tension in every case, tension going down two days after operation and remaining down from two to five weeks. The tension then returned to normal, but in no case did the tension go above normal after operation. He reported operating on ten eyes, or at least made the operation ten times on eight different humans in 1891.

In every case the tension was permanently reduced, according to his story, except in one in which the tension went up after operation. Dr. Barkan said, if I understood correctly, that there was no specifications as to what sort of cases the operation should be done on. De Vincentiis advocated operating on early glaucoma cases. Taylor suggested that it was a proper operation for other types of glaucoma. He spoke especially of hemorrhagic glaucoma and for glaucoma that obtained in cases with far advanced sclerosis of the retinal vessels. The idea was that this operation could be performed without the sudden reduction of tension and that there was no danger from the operation of intra-ocular hemorrhage.

He spoke of applying the operation to secondary glaucoma and also to inflammatory glaucoma. He also spoke of freedom from trauma and from danger of the operation, saying that we had a means of permanently reducing the tension with almost no danger to the eye. In 1900, nine years after the operation apparently had been done through these years, Scalina reported on 13 cases of hydrophthalmos — buphthalmos. He reported the permanent reduction of tension in these cases and success without event, except in one case in making the incision in a buphthalmic eye with a thin wall he went through the wall at the globe. There was a prolapse of the iris at that point. It is the only accident that is reported by any of these men.

A number of other men reported on the operation. De Wecker gave quite a dissertation on it and recommended it for a number of things, including myopia.

I do not want to prolong the discussion, but I should like to say again that the operation is easy of performance. Dr. Barkan's cases, some of them, have had normalized tension for a period long enough so that I think the operation should be used for trial.

Of course, the operation opens up many lines of speculation, which I shall steer clear of, but I should like to add this, if Dr. Barkan's operation does not have widespread approval and widespread adoption, I think Dr.

Barkan has done for us a very valuable thing in arousing our interest in the examination of the drainage angle. I think it is absurd that all these years when we have had means of examining the angle that we have not done it. When I say that we have not done it, I mean almost literally that. Only a few men have made a practice of examining the drainage angle.

Also, I think one should get away from the dread of the examination and look at it in this way, that the contact glass with illumination makes the angle visible for us. It gives us a view of the angle and that magnification helps. One can choose his type of illumination. He can choose his type of magnification, but think of it as a possibility through the use of the contact glass and then one can use whatever frills he wishes to use.

Dr. M. Uribe Troncoso, New York: Dr. Barkan's paper is an interesting and valuable contribution to the field of gonioscopy. His method is a modification of the original Koeppe's method of goniomicroscopy of the living eye; but considerably improved, as the patient is lying down instead of sitting and both the microscope and the slit-lamp are independent and moveable upon floor stands. The microscope may also be used hanging from the ceiling. This enables the operator to make finer adjustments for observation and relieves him of the heavy weight of the microscope held in the hands, as Thorburn, Werner, Castroviejo and I have done before.

Dr. Barkan uses the old contact glass with a cavity; I should prefer the new gonioscopic, more convex glass, which has an unobstructed view and gives better images.

Dr. Barkan's description of the angle in the living eye is fundamentally alike to Salzmann's and my own. There are, however, some differences which depend on the intensity of his illumination and the use of magnifications as high as 40 D. With the gonioscope I use from 15 to 23 D. only. Larger magnifications give, as in the ordinary microscope, a smaller field and change in some respects the appearance and color of the structures. For instance, the color of the ciliary body band is changed, becoming brilliant and diaphanous, the darker or brownish coloration fading. Dr. Barkan does not mention this ciliary body band, which is so characteristic in the gonioscopic picture, as a definite zone, with a different color from the iris. I have been able to find the reason of these differences between small and high powers, working with the slit-lamp in specimens of normal eyes, according to my method of microanatomy of the eye.

To explain the pathogenesis of simple glaucoma, Dr. Barkan has revived the theory of Henderson (1908) that sclerosis of the sclero-corneal or sulcus trabeculum, is the primary cause of hypertension. He also blames the deposition of pigment into the meshes of the trabeculum and even a pigment block of Schlemm's canal, as the responsible factor in glaucoma. In regard to sclerosis of the trabeculum, it seems difficult to me to make an accurate diagnosis with the slit-lamp. Even with the microscope and much higher magnifications, the detection of sclerotic changes is rather difficult. Dr. Verhoeff, who, in 1912, studied 10 cases of sclerosis of the trabeculum, found it to be not a primitive change, but a result of a previous peripheral synechia which had been detached. He defined the changes as

a thickening of the individual trabecula with narrowing or obliteration of the meshes and the development of connective tissue on the surface.

The obliteration of the opening of the trabeculum by pigment granules does not seem to me a frequent cause of chronic simple glaucoma. The deposition of pigment in the angle is usually a senile condition, and I have seen and made drawings of several eyes whose angles were permeated by pigment, in which no glaucomatous symptoms developed during a long period of observation. Other of these eyes were even hypotonic. Besides, we know that the canal of Schlemm is not the only outlet of the anterior chamber. The anterior surface of the iris has also a great absorbent power and contributes a great deal to maintain the balance of intra-ocular pressure. I have shown that even in cases in which the entire angle around the limbus is obliterated by anterior synechia, an iridectomy may lower permanently the tension without opening the angle itself.

An operation similar to the one Dr. Barkan performs was originally devised by De Vincentiis in 1893. He called it "incision of the iridic angle" and its aim was also to open and lay bare the canal of Schlemm from the inside of the anterior chamber, but, of course, at that time no gonioscopic examinations could be made, so that probably in many cases the incision was done over an already present peripheral synechia, with the result that the canal of Schlemm could not be reached. Besides, in many cases, the canal is obliterated in long-standing peripheral synechia. Probably for this reason the failures were in the majority, and the operation was abandoned.

Instead of a blind operation, Dr. Barkan is able by the gonioscopic method, to see the condition of the angle and operate only in cases where there is not peripheral synechia, that is mainly in cases of simple non-congestive glaucoma. In the congestive type, my statistics have shown that a peripheral synechia is a frequent occurrence.

The incision of the trabeculum and canal of Schlemm can undoubtedly be made accurately by observing the angle under the contact glass and microscope. It is, however, a difficult proposition to keep the air bubbles, which so hinder the view, out of the field. This operation should be tried with an open mind. Even if no sclerosis of the trabeculum is present it may be successful by providing an easier outflow of the aqueous from the anterior chamber to the intrascleral venous plexus and also changing the conditions of osmotic pressure in the anterior chamber.

Dr. Harry S. Gradle, Chicago: I would like to speak to one point of Dr. Barkan's paper, namely, the question of gonioscopy.

Unfortunately, I have not yet had the opportunity of practicing the operation of goniotomy. I am keeping an open mind as to the effects of the operation which can be determined only after sufficient time has elapsed to permit a good many men to try the operation and report end results after not a few weeks but a few years.

However, a great deal of credit is due Dr. Otto Barkan for reviving our interest in gonioscopy. I have been trying to see the chamber angle ever since Salzmann proposed indirect ophthalmoscopy. We followed the Koeppe method. We used the Koeppe device and the slit-lamp with a

great deal of trouble. It permitted us to examine the chamber angle at the inner aspect and with more difficulty at the outer. We obtained no information whatsoever about the chamber angle at the upper and lower margins of the limbus. However, with the present method we can obtain a great deal that is of importance.

The method of examination sounds rather formidable. In reality it is not so severe. I think it advisable for all who intend to practice gonioscopy to obtain an idea of what the chamber angle looks like under magnification of 15 to 20 diameters. But for practical use in gonioscopy methods that magnification is not necessary.

I have been practicing this since I have had the opportunity of spending some little time with Dr. Barkan recently. I found that for practical purposes the Koeppe contact glass and O'Brien hand slit-lamp for illumination and a three to five diameter binocular magnification suffices. I use the Bausch & Lomb, or Zeiss, head loop binocular, and I find that with three or five diameter magnification I obtain ample clinical evidence that is of great value.

When one practices gonioscopy with lower magnification the procedure is almost as simple as tonometry.

Unfortunately, in the majority of cases of acute uncompensated glaucoma where we wish to obtain accurate information as to the existence of adhesions in the chamber angle, it is impossible for us to see the chamber angle either because of the corneal edema, or because of the extreme shallowness of the anterior chamber. But, in the various forms of compensated glaucoma we can obtain clinical evidence that is of great value to us in determining the time and type of operation that may be indicated.

Again, I can recommend to you gonioscopy as a routine measure in your cases of glaucoma, particularly of the compensated type. I believe that for the next few years we are going to hear a great deal more of this feature.

Sir John Herbert Parsons, London, Eng.: I am going to make a comparison here on something that is admittedly not quite germane to the subject. At the Amsterdam International Congress I had the honor of meeting a dear old gentleman and a dear old lady. They were the father and mother of Dr. Otto Barkan. The father told me that in his early days he used to think that all of the gold was in California. I have been in the United States for some time and I have understood that all of the gold of the world is accumulated in the United States, but I have not seen any gold. But, so much for that.

With regard to the immediate question of Dr. Barkan's operation, I personally think it is a very great advance. I think there is a group of chronic glaucoma cases in which you have adhesion of the iris or a blocking of the anterior chamber by pigmentation, or a vascularization of the trabeculum, or you have cases of a similar nature which are the actual and fundamental cause of this condition owing to the interference of filtration. I think that is the explanation of the failure in most cases of the old iridectomy operation. Dr. William Langdon went so far in a case of chronic glaucoma as to almost recommend, and I think almost practicing in

many cases, iridectomy in the other eye in order to prevent the other eye becoming glaucomatous.

There is a good deal to be said on that. I think the failure of iridectomy is very largely due to the fact that iridectomy had the effect of seeing such an angle as is already opened up and tore off what I call the fourth angle, still leaving an adhesion and practically doing extremely little that was good.

I have already said in my address here that I regard trephine operations and most of the filtration operations as extremely crude methods of dealing with this problem. I would like to say, however, that I do not think that all cases of glaucoma are this type at all. I think there are cases of glaucoma in which there is probable blockage of the anterior angle of the anterior chamber, but the cause is such that the mere opening of the anterior chamber by any mechanical means is probably doomed to failure. We have to get further down into the root of the matter.

I say that the ordinary operations which are now adopted are, to my mind, extremely crude but that they have a pragmatic sanction in that they are successful in preserving sight in a large portion of cases.

The reason why I regard this as a step forward is that one is in a much more accurate way attacking the immediate cause of the disturbance. I think that the operation, even in cases which are otherwise suitable, is doomed to failure in a very large proportion of cases. I would like to look forward with hope to those results not being considered any more than the very early results which have been mentioned and as producing sufficiently pessimistic ideas as to prevent continued perseverence to the operation.

Obviously, it is a more delicate method and more accurate way of getting, if our theories are accurate, at the real sources of trouble. We are relieving that source by a more satisfactory method.

As Dr. Wheeler said, I think it is extremely palpable that it does not in every way afford a satisfaction that is going to reproduce the physiological conditions. If one comes to think of it, what a surgical operation does, every surgical operation is the relief of a pathological condition. In nine cases out of ten the satisfactory results in so far as in our case the eye and its vision is concerned, in the cases of many other operations in so far as the life of the individual is concerned — but, as a physiological condition, that is a thing which is almost beyond the scope of surgery. At any rate, the vast majority of cases have proven so.

I do not think that that must be regarded as a bar. I would urge that Dr. Barkan go on with these operations and that others should go out who are competent to deal with that type of extremely delicate operation, and should undertake them.

I must say that as my operating days are drawing to a close I feel some satisfaction that I have not to undertake such extremely delicate operations.

Dr. Otto Barkan, closing: I wish to express my sincere thanks to the discussers for their contribution to this subject. I will limit my remarks to but a few of the points brought out.

Replying to Dr. Wheeler, I should like to say that I have intentionally made the indications for this operation rather narrow at this time. I believe that the indications can be broadened later to include cases with peripheral adhesions of the iris. I have also successfully performed the operation in cases of secondary glaucoma and operated on one case of hydrophthalmos six months ago with normalization of tension over this six months' period.

In regard to the knife, I agree that a discission knife could be used provided its configuration is such that the anterior chamber is not lost. Experience, however, leads me to believe that my short-bladed goniotomy knife with the straight shank is preferable. Regarding the reference to Dr. Ugo Taylor, I included him as the assistant to De Vincentiis when I mention the latter's name. Reference to Taylor will be found in my previous paper, which is appearing in the Journal of Ophthalmology within the next few weeks and in the bibliography of today's paper. De Vincentiis, and the other Italians, operated without vision. They had no definite indications for they operated in every type of glaucoma. As Dr. Wheeler said, they included early and late glaucoma, absolute glaucoma, hydrophthalmos, inflammatory glaucoma; in fact, every kind of increased intra-ocular tension.

Concerning the novelty of my operation, it seems to me that it involves a completely new principle in the surgery of glaucoma in that a hitherto hidden area of the inside of the eyeball is made visible during the operation.

I wish to thank Sir John Parsons most especially for his remarks. It was incorrect to use the term "physiological restoration." I used it in order to convey the idea that with this operation we are restoring the physiologic direction of outflow in contradistinction to the abnormal pathways in external drainage operations.

I appreciate the valuable contributions to the discussion made by Dr. Troncoso and Dr. Gradle and thank you all for your very kind attention. I hope that this paper and discussion will open a wider interest in this subject.

VISUAL SEQUELÆ FROM EPIDEMIC MENINGOCOCCUS MENINGITIS*

PARKER HEATH, M.D.

DETROIT

PUBLISHED reports of complications resulting from meningococcus meningitis infections vary greatly. Uhthoff[1] (1906) found ocular complications in about 45%; Sinclair[2] (1919), 14.8%; Slesinger[3] (1933) found 9.5% visual sequelæ in children, and Kotliarewskaya[4] (1933) found 65.3%; Lazar[5] (1933), 12.4%, VI nerve paralysis, endopanophthalmitis. Dunphy's[6] review comments on the different parts of the eye affected in different epidemics.

TABLE 1

	UHTHOFF 1906	SINCLAIR 1919	SLESINGER 1933	KOTLIAREWSKAYA 1933	LEWIS[7] 1933	LAZAR 1933
Optic Neuritis	18			2	7	
Extraocular Paralysis	16	8	2	10		11
Pupil Anomalies	12			14		
Conjunctivitis		2		6		
Corneal Ulceration		2		1		
Metastatic Ophy. and Endophthalmitis	4	7	2	3		22
Blind and Normal Fundi				13	3	
Optic Atrophy					4	
Ages	Mixed	Mixed	Inf. to 15	Inf. to 11	Mixed	Mixed
Total Cases	110	128	42	75	184	266
Percentage Eye Complications Reported	45.4	14.8	9.5	65.3	7.6	12.4

* From Herman Kiefer Hospital.

TABLE 2 [8]

Meningococcus Meningitis, 1927-1931
Cases, Deaths, and Case Fatality, by Age Groups

AGE	CASES	DEATHS	DEATHS PER 100 CASES
Under 1	44	23	52.3
1	49	24	49.0
2	54	12	22.2
3	54	18	33.3
4	61	17	27.9
5	48	13	27.1
6	37	11	29.7
7	30	9	30.0
8	30	4	13.3
9	30	8	26.7
10-14	137	38	27.7
15-19	145	53	36.6
20-29	212	91	42.9
30-39	101	53	52.5
40-49	41	22	53.7
50-59	14	9	64.3
60	3	2	66.7
Totals	1090	407	37.3

Variations depend upon the form of treatment, immunity, strain of organism, site of process, standards of observations and interests, and reaction of different ages. Especially important is whether the observations were made during the acute states of the disease and convalescence, or after presumed recovery.

This clinical study was undertaken to determine clinical residue persisting in epidemic meningococcus infection, especially relating to the visual system and to compare these with other complications — particularly aural. The average interval between acute illness and these observations was 25.9 months with 68 selected cases, and 82.68 months with 25 unselected. Average ages and sexes were selected from the Detroit epidemic of 1928-31. At time of infection average age was 10.6 years. General,* neurological, ophthalmic and otologic examinations were made of 68 cases, showing a high percentage of complication. An additional

* General examination by Drs. John Gordon and F. H. Top; neurological by Dr. Ward W. Harryman; otologic by Drs. J. M. Robb and Hayden Palmer.

group unselected of 25 cases was used to compare the relation of seeing with hearing defects; average age 11.52 years; average interval 6.89 years.

The Detroit epidemic began in the spring of 1928 and reached a peak in May, 1929. The attack rate was increased from 3.1 per 100,000 population (1923-27) to a high of 60.7 per 100,000 in 1929, with a mortality of 30 deaths per 100,000 population. The age and death distribution 1927-31 is shown in Table 2.

The fatality rate for all ages was 37.3%. The organism was largely Type III. Specific treatment consisted of meningococcus serum given by intraspinal, intravenous, intraventrical and intracisterna magna.[8] The antitoxin of Ferry[9] had not been developed.

The epidemic disease begins as a nasopharyngitis (probably at times by a conjunctivitis), and by the blood stream route develops into a sudden, overwhelming infection as a meningococcus septicemia. The clinical complications during the early stages of the disease are those referable to the brain, cord, ear, eye, kidneys, joints (as arthritis), heart, skin and lungs (usually lobular pneumonia).

Clinical notes during the acute phases of the disease, in those cases reported herewith, mention the following complications: Petechiæ, herpes, abscess, serum sickness, lymphadenitis, tonsillitis and pharyngitis, conjunctivitis, arthritis, otitis media, squint, nephrosis, uveitis, exudative choroiditis, endocarditis, functional myocardial insufficiency, encephalitis, vaginitis, erysipelas, corneal ulcer, optic atrophy and syphilis.

The eye complications noted in the Detroit epidemic were endophthalmitis, panophthalmitis, iritis, iridocyclitis, uveitis, choroiditis, acute conjunctivitis, optic atrophy and strabismus.

Ocular muscle complications vary with site of lesion due to toxins and focus. The disastrous endophthalmitis characteristically begins early in the meningococcus infection; it is comparatively painless; slight congestion is present, and a grayish exudate, usually from the ciliary body, is seen, soon to appear in the anterior chamber. The tension is lowered. The hypopyon clears and the tension remains low, while vitreous changes persist and lead to the late chapters of "pseudo-glioma" and the blind shrunken eye. An early case reported with specimen by Mayou,[10] showed a thrombus in the ciliary body with exudation largely in this region. From a late enucleation (6 months after infection) Hayden[11] described the vitreous abscess as resulting from a septic embolus in the retina.

The following outline was used in general and neurological examinations: History, nutrition and body type; head size and shape; facial expressions; development; speech, tone, articulation, aphasia; mentality, consciousness, disposition, intelligence, ideas, emotions; posture, gait, station; atrophy or deformity; abnormal movements or tremor; coordination; paralysis; pupils; taste; smell; hearing; sensations, tactile, pain, motor position, vibratory, wrist and ankle, tendoachillis; biceps; triceps; knee; plantar; umbilical, upper and lower; cremasteric; cranial nerves; heart; lungs; abdomen; lymph glands; endocrine; joints; temperature; pulse; respiration; hemoglobin; white blood cells; blood pressure, and other special details and additions.

The general medical examination showed the following in the selected 68 cases:

Enlarged liver	6 cases
Enlarged heart	4 cases
Mitral endocarditis	1 case
Functional myocarditis	2 cases
Enlarged spleen	5 cases
Markedly enlarged suboccipital glands	2 cases
Cervical marked gland enlargement	1 case
Dyspituitarism	1 case

Two cases were syphilitic, neither in the group numbered above. Blood pressure, blood hemoglobin and white cell counts were made from many without unusual findings.

Neurological examinations of the complicated series (apart from eye and ear) showed the following sequelæ:

Pseudo-hypertrophic muscular atrophy	1 case
Encephalitis	3 cases
Hemiplegia	1 case
Marked vasomotor changes	2 cases*
Tremors	2 cases
Cerebellar ataxia	2 cases
Sensory residue upper extremities	1 case
Facial palsy	1 case
Very unstable emotional tone	2 cases
Disturbances in gait	4 cases†
Personality changes	10 cases

* 1 also with markedly dilated pupils.
† Foot eversion 1, right lower leg only 1.

The last item is a comparison of the patient's reaction to his environment after meningococcus infection with that before. Excessive irritability, temper outbursts, apprehensions, juvenile fears, sadism and other sharp differences were used to establish this group.

In the selected group of 68 cases deafness was found as follows: Bilateral, 20; unilateral, 4, associated with labyrinthine disease. Hearing loss was measured by voice, fork, audiometer and caloric tests and was found to be partial in a fair proportion of cases. A separate series of 85 cases showed: Partial unilateral deafness, 7; bilateral partial, 4; complete unilateral, 5; complete bilateral, 14 examples. With a selected high percentage of deafness there was found an increase, but not a parallel increase, in eye complications.

TABLE 3

Ocular Residue from 93 cases Epidemic Meningitis
Interval average of 54.29 Months

MUSCLES AND SQUINT		PUPIL CHANGES	ENDOPHTHALMITIS	FUNDI
		Both dilated		Optic neuritis and edema
Convergent squint...	2	1	O.D. 2	1
		Anisocoria		
L. sup. rectus palsy and head tilt	1	1	O.S. 1 O.U. 1	
				Neuro-retinitis in one eye, associated with phthisis-bulbi stage of endophthalmitis
Nystagmus intermittent including vertical..	2			1
Divergent	1			
Convergent weakness	1			
Total	7	2	4	2
Percentage	7.5	2.15	4.3	2.15

The eye residue which may be ascribed to meningococcus infection listed in Table No. 3 total 16.1%, and was 22% in the high complication group.

Muscular disturbances seen early in the disease disappear. Other muscle findings were two convergent and one divergent squint, one left superior rectus defect and one loss of convergent power. The two examples of nystagmus were not constant nor in all directions of gaze, but each was a definite vertical type on upward gaze and not associated with low vision.

Pupil changes were abnormal dilatation with diminished range of reaction to light, associated with marked vasomotor disturbances in all the extremities; the other case showed persistent unequal pupils apparently acquired during the disease, the light and accommodation reactions were present. The all or none character of endophthalmitis make this complication consistent early and late in the disease. The reported cases agree fairly well in percentages. The writer has not seen a case showing the vitreous stage of exudation recover vision. The use of Ferry antitoxin, as reported by Hoyne[12] and Lazar,[5] offers hope both to prevent this complication and also to stop such a process under way. Because cases of pseudo-glioma without history of meningitis in which the meningococcus was recovered from enucleated eyes have been reported,[13] one must bear this agent in mind in considering etiological factors. An example of an optic neuritis, with edema on the nerve heads of over one diopter, associated with chronic photophobia, was found; and one eye with neuroretinitis, the other shrunken from endophthalmos. The other fundus changes were not thought to be aftermaths of meningococcus.

Summary

Medical, neurological, ophthalmic and otologic examinations were made of 68 cases selected because of high proportion of complication, 25.9 months after epidemic meningococcus infection. Ophthalmic and otologic examinations were made of 25 additional unselected cases 82.68 months after the epidemic. The medical examinations showed enlarged livers, spleens and hearts in from 5.8% to 8.8% of the 68 cases; other complications persisting were comparatively infrequent. The neurological manifestations remaining after the acute disease were especially pronounced and severe in the personality field. These increased with the selected increase of other complications. The visual sequelæ averaged 16.1% in 93 cases and were divided into muscle disturbances, 7.5%; pupil, 2.15%; endophthalmitis, 4.3%, and fundus changes, 2.15%. Many eye complications in the acute state

of the disease were fugitive, but those persisting apparently had been acquired early in the disease. Acute disease is composed of causative agent with its various secondary side actions. The late or chronic disease is often a picture of the side actions alone. Loss of hearing, total and partial, was a most frequent and serious complication, and in these selected cases the visual sequelæ increased but not proportionately with the auricular. The actual percentage of permanent visual complication sequel to the disease must vary in different epidemics and different parts of the same epidemic. Allowing for the higher proportion of complication in those cases returning to the hospital due to their availability, visual residue is less than 10%. One is led to the conclusion that most of the visual damage in this disease is done early during the overwhelming septicemia and toxemia, and that a continuing process is relatively uncommon. This is a hint for early and full therapeutic efforts. However, as shown by Wertham,[14] deep lesions occur in the nervous parenchyma, cortex chiefly, but also in subcortical structures. The neurological findings reported herein substantiate this, and suggest a continuing process.

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DISCUSSION

Dr. Wells P. Eagleton, Newark, N. J.: Dr. Heath's paper is a valuable contribution for he has carefully analyzed a large number of cases and shows the frequency of the different ocular sequelæ; and all I can add is to compare the pathological lesions of meningococcic meningitis which are known to cause its symptoms and sequelæ, with the lesions of the ordinary pyogenic meningitis from a suppurating ear or nasal sinus.

In dissecting injected heads,[1] I noticed two anatomical facts to which my attention has never been directed that have been of first importance to me in the early diagnosis and treatment as well as in understanding the sequelæ of the ordinary form of meningitis.

(1) All the sensory cranial nerves have a prolongation of arachnoid surrounding them for some distance after leaving the dura, so that the anatomist can immediately recognize its sensory character by this arachnoid protection. On the other hand the motor cranial nerves have no arachnoid prolongation, losing their covering immediately on entering the dura. This explains how slight displacement of the brain is apt to cause paralysis, the motor sixth, third and fourth nerves, while all sensory nerves are spared.

However it is in the subarachnoid prolongations of the basal cisterns surrounding the sensory nerves that accumulations of exudate lodge, and are the cause of many localizing symptoms, and when firmly organized cause the sequelæ of pyogenic meningitis.

Again, in trying to fill the subarachnoid system from the inner ear (after having knocked away the outer labyrinthine wall) by filling the inner ear with a colored fluid, and allowing it to flow by gravity, I found the communications between the arachnoid spaces leading from the peripheral prolongations to be so small that a fluid dye which contains but small solid particles could not pass, while liquid solution would. Obliteration by adhesion of these fine arachnoid passages into cisterns which are adjacent to the infected area is the mechanism by which the spread of infections to all parts of the system is prevented in the usual types of pyogenic meningitis that come from the ear or the nose. It should be emphasized that the germ of epidemic cerebrospinal meningitis acts differently from any of the other pus producing germs. Meningococcus not only is (a) not of great virulence; (b) "It has comparatively little surviving power when brought into contact with mesodermal tissues, although (c) it can live for some time in relation to ectodermal cells."[2] And (d) meningococcic infection is a meningococcic sepsis with secondary meningeal localization[3] (Herrick).

Thus in all other pyogenic meningeal infections, streptococcus, pneumococcus, and staphylococcus from a local focus of infection, the blood stream is sterile until the terminal stage, that is when the hemocerebrospinal fluid barrier between the blood vessels and the arachnoid mesh breaks down, and allows the germs to enter the blood stream, at which time the patient has a meningo encephalitis, from which, in my experience, none recover.

However, in meningococcic infections, the blood stream is attacked before the arachnoid spaces, and the cranial nerves are not attacked by pressure but the vascular nature of the infection may act directly on small vessels of the ependyma on the cerebral tissue or nerves by embolic infarcts or by its toxins[4] (Wertham).

Thus the ocular motor paralysis is probably not due to pressure from the exudate, but to changes in the cerebral tissue or nuclei.

The meningococcus stands in its virulence and mode of invasion midway between the pyogenic germs, such as the streptococcus and pneumococcus, and the neurotropic viruses, such as poliomyelitis and encephalitis.

At the Essex County (N. J.) Isolation Hospital, in the vast majority of cases, the blood stream has become sterile before the diagnosis of meningococcic meningitis has been made by a lumbar puncture, while in a few cases the meningococcus was still to be isolated from the nasal secretions.

The optic nerve is involved as a perineurositis.[5] There is not a papilloedema in contradistinction to that papilloedema from pressure that occurs when the optic prolongation of the third ventricle presses on the chiasm in meningitis of pyogenic origin.

Again *impaired hearing* in pyogenic meningitis is due to effusions in the arachnoid space; the deafness is never complete because it is due to pressure on the eighth nerve, which withstands pressure very well. On the other hand, the deafness of meningococcic meningitis is apt to be complete because the lesion attacks the nerve tissue itself, either in the labyrinth or brain.

In the protective form of exudate in the posterior fossa, as occurs in labyrinthitis or skull fractures, the time of the vestibular reaction to turning is reduced to about 50% — and this continues for all time, as I pointed out in 1922[6] — but there are no symptoms at the time of invasion; however, in the embolic process of meningococcic infection there is nystagmus for some time.

I wish to congratulate Dr. Heath on his paper, and taking his findings as a basis, I am going to suggest that the serologist at least attempt to prevent some of the ocular sequelæ by treatment of the various lesions causing the ocular disturbances by injections of serum at the site of the local embolic deposit or neurological manifestation, the same as we are attacking these areas in the treatment of surgical meningitis.

I hope each individual examination on which Dr. Heath has compiled his statistics will be made available to all students of meningitis, as it is only by a careful analysis of individual cases that any progress in the treatment of meningitis is made possible.

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DR. PHIL M. LEWIS, Memphis: Dr. Heath has done a very difficult thing in following so many of these patients for over three years. I know by experience how hard it is to keep in touch with them.

It has been my good fortune to have examined the eyes and recorded the findings in over 300 cases of meningococcic meningitis.

During an epidemic, the percentage of eye complications is higher than it is in sporadic cases. About 50 per cent show some departure from

normal. Any or all structures of the eye may be involved. The three principal complications are optic neuritis, strabismus and endophthalmitis. Minor complications are conjunctivitis, rarely keratitis, pupillary abnormalities and vascular changes. Amaurosis occurs only rarely, but is quite serious.

All ocular complications of meningitis tend to recover except endophthalmitis. Conjunctivitis and other minor conditions clear up easily. Optic neuritis, if mild, recovers practically completely with good central and peripheral vision. Severe cases are always followed by various degrees of optic nerve atrophy, corresponding contractions of the visual fields and often considerable reduction in central vision. Strabismus, which is practically always paralytic and usually of the sixth nerve, nearly always recovers. Endophthalmitis almost always ends in complete blindness because of the extensive involvement, often with complete retinal detachment. (Lantern slides shown).

DR. PARKER HEATH, Detroit: I want to thank Drs. Eagleton and Lewis for their discussions.

One part of the study not read concerns certain animal experimentation. The speaker had the privilege of observing the monkeys by means of which Ferry developed his antitoxin. There were about 50 of these monkeys and not a single one showed endophthalmitis. Two or three showed ptosis. There were no fundus lesions seen. Some became obviously blind. No doubt the blindness was due to a central lesion. The sections of the brains and cords showed scattered deep lesions. The disease is not to be looked upon as a pure meningitis, ordinarily speaking; it really is a deep brain process frequently.

NODULAR DYSTROPHIES OF THE CORNEA

BENJAMIN RONES, M.D.

WASHINGTON, D. C.

THE CORNEAL dystrophies still remain an ophthalmological jig-saw puzzle even though Groenouw[1] first called attention to them in 1890. Since that time several varieties have been separated clinically, although no progress has been made concerning their etiology and very little with reference to therapy. Their pathology also presents a picture of confusion which has not yet been correlated.

Groenouw first described this condition (Knötchenformige Hornhauttrübungen) as consisting of numerous small, roundish, gray, non-confluent opacities in an otherwise clear cornea. These tend to involve the central portions, leaving the periphery clear. Also, the larger nodules bulge the epithelium forward, causing it to be very irregular. These opacities remain unchanged for many years, usually without inflammatory changes. Both eyes are always affected, and it sometimes occurs in several members of the same family.

The type of nodular dystrophy described by Salzmann[2] differs from the above in that the nodules are larger and less numerous, with marked elevation above the surface of the cornea. It occurs usually in women and is not familial. Most of the reported cases are unilateral and give a history of repeated attacks of eczematous kerato-conjunctivitis. Salzmann feels that this variety of dystrophy is not as rare as the paucity of reported cases leads one to believe.

The lattice type of corneal dystrophy was first reported by Haab,[3] who described it as "alphabet keratitis," beginning at puberty and always being bilateral. Here the opacity consisted of many fine lines arranged in a lattice-like pattern. Dimmer[4] felt that recurrent inflammation was present early in the condition, but Parsons[5] did not concur in this opinion.

The case to be reported is to my mind a typical one of Groenouw's nodular dystrophy, as distinct from the other two types.

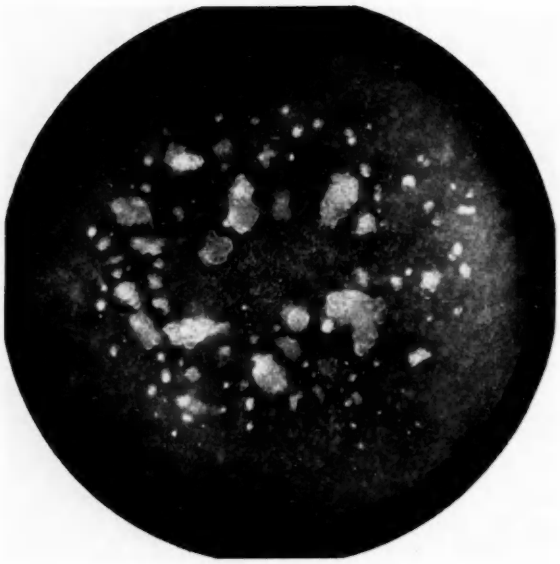

FIG. 1 — (Rones) — Nodular Dystrophy of Cornea, Right Eye.

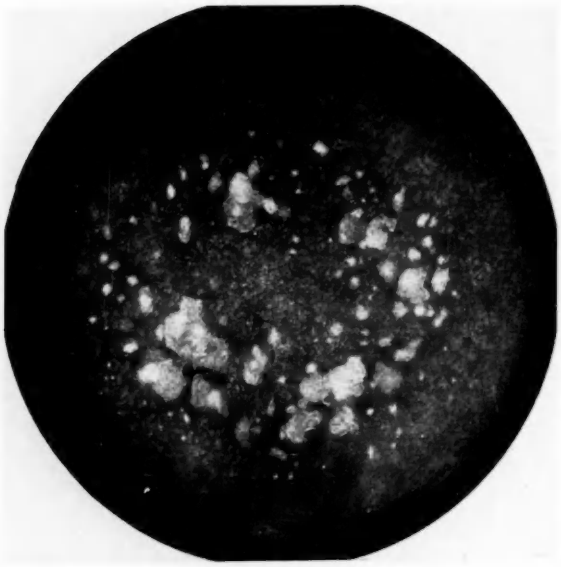

FIG. 2 — (Rones) — Nodular Dystrophy of Cornea, Left Eye.

Miss M. R., age 26, a secretary by occupation, was first seen on September 20, 1935.

F. H. — There is no history of ocular disease in the family, and she is positive that no other members have a condition similar to her own.

P. H. — During childhood had whooping cough, measles, mumps, and scarlet fever. Since then her general health has been very good.

P. I. — Four years ago she first noticed slight haziness of vision. Her ophthalmologist found corneal opacities in both eyes, with 20/30 corrected vision. He used dionin and sodium iodide and though he noted no change in the appearance of the corneæ, the vision improved to 20/20 in each eye. Recently she has again noticed some slight haziness and failure of vision. At no time has there been any redness or irritative symptoms.

EXAMINATION OF THE EYES:

Externally: The lids are negative. Palpebral conjunctivæ are smooth and glistening, and there is no hyperemia of the bulbar conjunctivæ. Both corneæ show opacities of varying size, scattered over a peri-central zone, leaving the periphery clear. The tactile corneal reflexes are normal. The pupils are equal and regular, and react actively to light. Tension to fingers is normal.

Ophthalmoscopically (with dilated pupils): Lenses are clear. No vitreous opacities. Discs are of normal color and outline, with physiologic cupping. The vessels are of normal appearance. No lesions are visible either in the macular or peripheral fundi.

Slit Lamp: Both corneæ show essentially the same picture. The opacities are of a grayish-white color, varying in size from pin-point to 3-4 mm. They are sharply defined, although a few of the larger ones are confluent. The center of each cornea is quite clear, with only a comparatively few minute scattered opacities. The others are ranged in a zone surrounding the center. There is no bulging forward of any of the opacities, and the overlying epithelium has a smooth healthy appearance and takes no color when stained with fluorescein. In a narrow beam most of the opacities can be localized in the anterior third of the corneal stroma though some of the larger ones extend deeper, even to Descemet's membrane. There are no cellular deposits on the

FIG. 3 — (Rones) — Slit Lamp Appearance of Nodules.

endothelium. The iris patterns are sharply defined, with no evidence of uveal disturbances. The aqueous ray is not visible.

R.E.V. = 20/40 — 2; w c — 1.25 × 150° = 20/40
L.E.V. = 20/50; w s — 0.50 = c — 1.25 × 40° = 20/40

Reads Jaeger 1 at 10 inches with each eye.

Fields: Normal for form and color.

PHYSICAL EXAMINATION:

General examination negative. Blood pressure 120/90. Urine negative. R.B.C. 5,010,000. W.B.C. 4,250, with normal differential. Blood Wassermann negative. Intradermal tuberculin test negative even to 1 mgm.

The clinical appearance and the non-inflammatory course of this case leads me to classify it in the Groenouw type of nodular dystrophy. This brings up the question, however, as to the validity of the classification of the dystrophies. Wherein do they differ, other than their clinical appearance, and are they not simply a variegation of the same disturbance?

If one examines the pathological reports of these conditions, one finds a wide variety of findings. Groenouw,[6] studying super-

ficial scrapings, found hyaline deposits, staining deeply with eosin. Fuchs[7] trephined such a cornea and found that the changes were limited to the superficial layers. There was an edema of the superficial lamellæ, with raised spots which corresponded to the larger opacities. In places where the changes were most advanced, amorphous masses lay between the swollen and the normal lamellæ. These gave no mucin reaction when stained with thionin. Also the superficial layers of the lamellæ, which seemed normal when stained with hæmatoxylin and eosin, showed changes with other stains. They were colored violet instead of blue by thionin, pointing to a mucoid degeneration which was lost in the later stages. On the basis of staining reactions Fuchs discriminated between an acidophilic (red) substance which was found in masses or layers on Bowman's membrane, and a basophilic (blue) substance found in masses and as a diffuse invader of the lamellæ. Goar[8] was able to demonstrate amorphous masses resembling hyaline, and staining yellow with trinitrophenol.

Brown and Katz[9] reported a case of the Salzmann type of dystrophy, in which they excised three nodules and examined them histologically. The pathological picture in all of these nodules varied considerably and the authors felt this was due to their being in various stages of development. From this pathologic picture they concluded that the condition was a non-inflammatory, slowly progressive process, manifesting early hypertrophic changes going on to a resultant degenerative picture. There were marked and various alterations of the epithelial layer, Bowman's membrane, and the outer third to one-half of the corneal stroma. Brown and Katz felt that the pathological reports of Salzmann and Ladekarl,[10] which differed from each other and from their own case, could also be explained by this variation in the stage of the condition. It was their opinion that Salzmann's nodular corneal dystrophy could be diagnosed only by its clinical history and appearance and not by its pathologic changes.

In the lattice-type of dystrophy, Haab showed that a transparent substance was deposited on Bowman's membrane. Paderstein[11] felt that a degenerative process occurred in the epithelium in which the nuclei were destroyed and the protoplasm changed into a hyaline substance accumulating on Bowman's membrane. Wirth[12] also noted such a hyaline substance. Löwenstein[13] examined small corneal sections and found acidophilic deposits on Bowman's membrane and basophilic granular deposits just beneath it.

In Chou's[14] case, Bowman's membrane was not recognizable and the superficial lamellæ which stained a deep pink appeared more or less homogeneous. He could find no evidence of mass-like accumulations between the lamellæ.

Maury[15] recently reported a case showing the clinical features of both the nodular and lattice dystrophy. He concluded that the primary deposit of hyalin material takes place in the corneal lamellæ themselves, and that the primary lesion is a lipoid degeneration of localized portions of the lamellæ.

We thus find that in these three conditions there is a rather wide variation in the type of pathologic process reported. A fairly common feature, however, is the report of the hyalin-like deposits in all three conditions, although there is dispute as to whether this is initiated in the epithelium or in the superficial corneal lamellæ. There has also been some controversy as to the connection of these lesions with the corneal nerves. Stanka,[16] by the use of supra-vital stains, could find no such relationship. Koby[17] believed that the condition was due to a chronic inflammation of the corneal nerves or to nodular swelling of the nerve endings. Collins[18] felt that the lesion began in the nerves and nerve endings and was, therefore, neurotrophic. Kraupa[19] and Löwenstein concurred in this opinion.

Numerous theories have been offered to explain the origin of these dystrophies. In reviewing the literature one is struck by the number of cases occurring in members of the same family. The most noteworthy of these is Frykholm's[20] series, where eighteen cases occurred in forty-two members of a family, over six generations. Judd[21] reported nine cases in three generations, and Freiberger[22] twelve cases in nine generations. On the basis of his series, Freiberger called the condition "familial corneal dystrophy." This term should not be adopted, for a large number of the reported cases, including my own, have been present in only one member of a family, with no history suggesting others being involved. The authors of the three series quoted above consider that the condition is inherited as a dominant trait, and it is not sex-linked. It occurred with equal frequency among the male and female members of the families and was transmitted through both sexes.

As was to be expected, the condition has been attributed to endocrine disturbance by several authors. Wirth reported a case,

in which evidences of hyperthyroidism were present, and consequently he considered the corneal condition resulted from the thyroid dysfunction. Freudenthal[23] believed that one of his cases showed evidence of hypothyroidism together with hypopituitarism.

Wehrli[24] suggested that the condition was an inactive form of corneal tuberculosis. The pathologic reports do not bear out this contention.

Fuchs considered that these dystrophies resulted from a nutritional disturbance, while Collins felt that they were neurotrophic in origin, although neither could offer much evidence in their support. It is my feeling that the probable solution lies in the combination of these theories, as supported by the recent work on the metabolism of the cornea.

Fischer[25] has demonstrated a close relationship between the transparency of the cornea and its water content. If the cornea loses or takes up more than about twenty to thirty per cent of its water content it becomes semi-translucent. He has also shown that the imbibition of water is related to the integrity of the epithelium and endothelium, and that if either layer is injured the cornea becomes permeable to water in both directions. He has observed that the intact cornea maintains an irreciprocal permeability towards water and sodium chloride, and that the unidirectional flow is towards the aqueous. However, Krause[26] feels that the epithelial permeability is evidently influenced through the action of the trigeminal nerve. Tagawa[27] has found that the water content of the epithelium was regulated by the nerves, and that after nerve irritation the amount of water in the cells was increased. Also after sectioning of the trigeminal nerve, the epithelium swelled and islands of cells were desquamated.

On the basis of this work one can offer the hypothesis that the initial lesion in the corneal dystrophies is an irritative lesion in the corneal nerves. This results in an increased permeability of the epithelium, with an increase in the water content of the epithelial cells and superficial lamellæ, and a change from an irreciprocal to a reciprocal corneal permeability. This brings about a disturbance in the oxygen metabolism, resulting in a necrosis and hyalinization of those cells which are farthest removed from the limbus and aqueous; that is, in the superficial layers of the center of the cornea, as is seen in this type of corneal dystrophy.

SUMMARY

1. A review of the literature of the corneal and lattice dystrophies leads to the belief that they are merely variations of the same condition.
2. Although the variety of the pathological findings is confusing, the frequency of hyalin masses in the epithelium and superficial lamellæ would allow it to be called the characteristic lesion.
3. The theories offered in explanation of these dystrophies are discussed and held to be insufficient.
4. An explanation is offered based on the disturbed permeability of the cornea resulting from an irritative lesion of the corneal nerves.

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DISCUSSION

Dr. Mark J. Schoenberg, New York: Dr. Rones' excellent paper has covered the subject so well that there is nothing to be added as far as the clinical picture of corneal dystrophies is concerned.

Considering the fact that this condition is frequently found in several members and several generations of the same family, at a certain age period, that in some cases it is associated with psychopathic manifestations, defects of intelligence, tower skull, etc., we are led to believe that the constitution-endocrine factor plays an important rôle in the origin of this pathologic entity. It seems to me that such cases will be better understood if the cooperation of men who devote special attention to endocrinology and constitutional aberrations would be secured.

Some years ago, Fuchs expressed the opinion that corneal dystrophies are due to nutritional disturbance, while Collins thought that they are of neurotrophic origin. To us, these opinions do not sound as weighty nowadays, because they are both one and the same thing. The question is: What is the cause of this neurotrophic or nutritional disturbance? Are we dealing with one of the abiotrophies, an endocrinopathy, or some other unknown factor? It would be idle to speculate on this subject before other data are cleared up.

Very stimulating and interesting is Dr. Rones' attempt to formulate a hypothesis as to the manner in which the nodular dystrophies of the cornea are formed. Fischer, Tagawa and Krause contend that the *imbibition* of water by the corneal tissue is related to the integrity of the epithelial and endothelial layer, that the *permeability* of the cornea is influenced by the action of the trigeminal nerve; that the *water content of the epithelium* is regulated by the nerves and that after nerve irritation, the amount of water in the cells was increased. Based on the findings of Fischer, Tagawa, and Krause, Dr. Rones advances the hypothesis that the initial lesion in the corneal dystrophy is an irritative lesion in the corneal nerves; which irritation results in an increased permeability of the epithelium with an increase of water content of the epithelial cells and superficial lamellæ. This is followed by a disturbance of the oxygen metabolism with formation of nodules of necrosis and hyalinization.

The theory is interesting, but like any other theory it will have to defend itself against a number of questions and objections:

1. Why does the dystrophy appear in nodules? Why not diffuse?
2. Why does it appear in *several members* of the *same family* and at times in several generations?
3. Why does it never appear in trigeminal neuritis?
4. Several years ago, I studied the corneal nerves by the aid of vital stains. One of my cases had a nodular dystrophy of the cornea. The vital stain showed no abnormality of the corneal nerves or nerve endings.
5. That that living cornea *does not behave like a sponge*, ready to imbibe a large quantity of water whenever the epithelial or endothelial layers are damaged, is to my mind proved by the following experience: I have been studying the corneal permeability in the living eye by injecting a stain under the conjunctiva adjoining the corneal limbus. Within a half

hour, the dye stained the periphery of the cornea and appeared in the aqueous. Since the fluid injected made its way from the subconjunctival space into the corneal tissue proper without having to pass through epithelium, one might have expected that the corneal tissue, which is so avid for water, would have retained not only the stain but would have imbibed water. This would be easily ascertained with the aid of the slit lamp microscope. But, nothing of the kind happened. The cornea did not swell up. In other words: in vivo, the corneal parenchyma is not behaving like a sponge towards water.

Although none of the above reasons are sufficiently important to obliterate Dr. Rones' hypothesis, they are sufficiently interesting to invite an answer.

May I repeat, Dr. Rones' paper is a valuable and stimulating piece of work and I am very grateful for having been allowed to discuss this paper.

SECONDARY CATARACT WITH PARTICULAR REFERENCE TO TRANSPARENT GLOBULAR BODIES*

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WILFRED E. FRY, M.D.

PHILADELPHIA

TRANSPARENT globular bodies frequently constitute a very interesting feature of after-cataract. A few of the many cases, as seen with the slit lamp and corneal microscope, are illustrated in Figures 1, 2, 3, 4, 5, and 6.

Little has been written about this peculiar and frequently occurring phenomenon, although J. Hirschberg, in his introduction in the Augenheilkunde (1901, S. 159), as quoted by Vogt,[1] remarked on "drops" which form not only a part of various cataracts but also are found in after-cataract membranes. Elschnig[2] described them exactly and made microscopic examination. He failed to mention Hirschberg.

Koby[3] says that transparent globules are found more frequently in the young than in the old, that rarely he met with globular cells after the fiftieth year. This has not been our experience. There seems to be no age preference. Of the 4 cases of after-cataracts with globule formation described by Vogt,[4] 3 were of patients aged 62, 63 and 64 years.

The globules are seen only after division of the lens capsule, never after all remains of the capsule have been removed. Visual acuity is affected, depending on the extent of their occupation of the pupillary area. They vary in size up to 2 mm. in diameter (Fig. 1) and are similar in appearance and action to perfectly transparent soap bubbles. Sometimes one or more can be seen hanging by a thin pedicle, but mostly they occur in bunches. When the globules are very small the mass appears like so much foam or froth. Not infrequently we have seen the whole pupil filled with them.

Whether a single globule or a large number is seen, they always extend from a portion of the remaining lens capsule. Some are spherical, others are ellipsoid. Often they fluctuate with the movements of the eye. They continually change in appearance.

From time to time the picture differs. In a surprisingly short time some globules disappear and new ones appear. They are produced rapidly, are seen a few days after operation and continue to form, vanish, and others form again, the process continuing for years. One is given the impression that they blow up and finally burst like a toy balloon. Sometimes, when the process is arrested, the globule wall gradually loses its transparency.

While the exact nature of these bodies is not understood and while they are more frequently seen after operation, they may occur after any separation of the capsule as in the case (Fig. 6) in which they were produced after a knife prick of the capsule. Vogt has seen them develop in the interior of the lens after contusion.[5]

In one instance, a woman 58 years of age, the whole pupillary space was filled with a layer of these globules. The entire membrane was removed by the method of Lyster,[6] and the microscopic appearance is illustrated in Figures 7 to 11, inclusive. The gross specimen consisted of a small, irregularly quadrilateral, grayish membrane. After fixation in 10 per cent formalin, it was transferred to 70 per cent alcohol and examined under glass by the slit lamp. When examined in vivo this tissue had exhibited innumerable, small, glistening, rounded globules; but after fixation, the greater number of these could not be seen. There was, however, near one margin of the material a small area in which there were still a few globules. These globules were small, but were typical of those seen in the eye previous to extraction of the secondary membrane. Therefore, it was considered advisable to section this membrane serially.

A typical section of the membrane formed an arc of tissue about 6 mm. in length, consisting chiefly of posterior lens capsule in the middle portion and equatorial and anterior lens capsule at either end. In many of the sections the anterior capsule was folded inward and there was included in the space so formed a number of degenerated lens fibers. The posterior lens capsule was thin and presented no noteworthy features except that in several

sections there was a longitudinal split and in others the anterior capsule, which was moderately thick, showed capsular epithelial cells on both sides. This appearance must have resulted from proliferation of the capsular epithelium around the torn edge of the anterior capsule. At times these cells exhibited marked changes. The cell wall was circular in appearance, the cytoplasm had disappeared, except for a few granules, and the nucleus was either flattened against the cell wall or was entirely absent. At other points there was marked proliferation of capsular epithelium so that the cells were four or five layers in depth. In the places in which there were such layers of epithelial cells, they were not piled on top of one another, but were separated by loose, interlacing bands of hyaline-like tissue, similar to lens capsule. These hyaline-like bands were in a position to be the product of an abnormal activity of the epithelial cells. In some places cross sections of lens fibers had the appearance of large, irregularly rounded spaces, filled with a fine, granular deposit — the so-called bladder cells. There were no spheres of Morgagni. No calcareous areas were noted, and no slits which might have contained cholesterin crystals were present.

The three features which are pertinent to the present discussion are: first, the vesicular or bladder cells; second, the interlacing bands of hyaline tissue; and third, the cystic appearance of a certain number of the proliferated and swollen capsular epithelial cells. The bladder cells probably do not account for the appearance of the globules described by Hirschberg and by Elschnig. These cells commonly are found within, and should be seen in most cataractous lenses. But no such formations were noted. Concerning the second feature, the interlacing hyaline bands, these had a folded appearance at places that suggested collapsed vesicles, but it seems unlikely that they could account for the delicate, walled structures under consideration. It seems most likely, therefore, that the globules of Hirschberg and Elschnig are formed from cystically enlarged capsular epithelial cells.

Clinically, it had been found that the occurrence of these globules on secondary cataractous membranes was so exceedingly frequent that it was thought advisable to examine sections of enucleated eyes containing secondary cataracts in order to determine whether any structures other than those found in our speci-

men could account for the formations seen by the slit lamp. Accordingly, a number of specimens from the collection of pathologic material at the University of Pennsylvania and at the Wills Hospital were examined. No additional information was obtained from this search.

The fact that intraocular cells can form large, thin-walled, vesicle-like structures is not limited to the capsular epithelium of a secondary cataract. Such a vesicle-like structure was seen in a lens, dislocated within the vitreous, but which, otherwise, was intact. The capsular epithelium of this lens showed that it was abnormal to a certain extent, in that it had proliferated so as to line the whole posterior capsule. This structure is seen in Figures 6 and 7. It is also known that similar processes occur in cells on the surface of degenerated retinas, as shown in Figures 8 and 9. The cellular derivation of such vesicles is clearly indicated in Figure 10, in which a flattened nucleus is still visible.

It is noteworthy that in Elschnig's description the histological examinations were made on the eyes of two individuals. In neither case is there an accompanying clinical or slit lamp description. The histological descriptions are essentially those of a secondary membranous cataract with a well marked Sommering ring. The hemispherical nodules which he describes are solid formations, part of which contain nuclei and part of which do not. He states that the red stained ones are without nuclei. This indicates to us that they have undergone greater degeneration. He does not distinguish these sharply from the well-known bladder cells.

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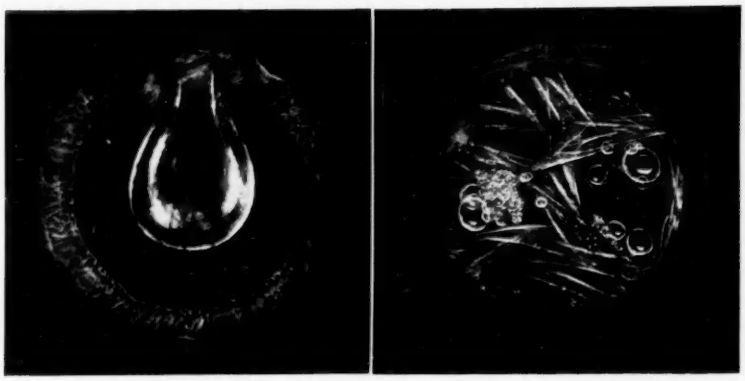

FIG. 1 FIG. 2

FIG. 1 — (Cowan and Fry) — Mrs. F. K., age 65, diabetes, arteriosclerosis. Drawing made 8 days after lens extraction. Six weeks later the large globule could not be seen, but there was a bunch of small ones in the upper pupillary space. These continued to change until the last time seen, 32 months after operation.

FIG. 2 — (Cowan and Fry) — Miss S. H., age 25. Drawing made 2 years after discission for congenital cataract.

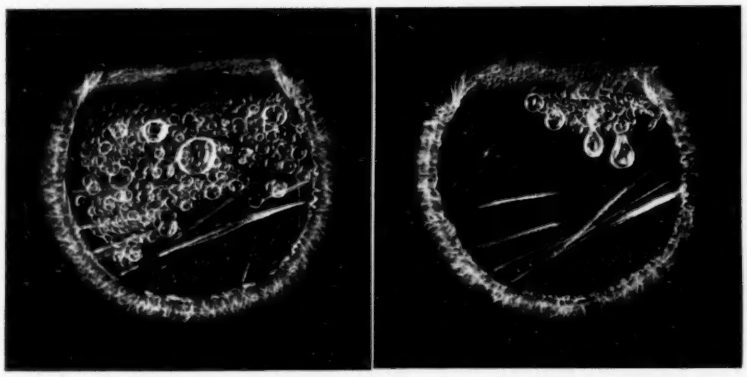

FIG. 3A FIG. 3B

FIG. 3A — (Cowan and Fry) — Mrs. E. M., age 58. Drawing made 2 years after combined extraction of senile cataract. Hernia of vitreous below lower edge of remaining capsule. Vision 6/20 with correction. Condition continually changed.

FIG. 3B — (Cowan and Fry) — The same eye 2 years later, still changing.

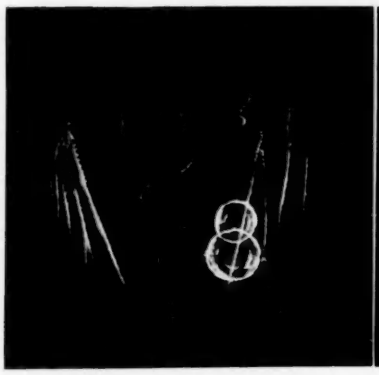

FIG. 4A

FIG. 4B

FIG. 4A — (Cowan and Fry) — J. D., age 8. Drawing made 70 days after linear extraction and iridectomy for traumatic cataract. Ten days ago only one globule was seen.

FIG. 4B — (Cowan and Fry) — Same eye one month later. The globules increased in number until 4 months after operation pupil was entirely filled.

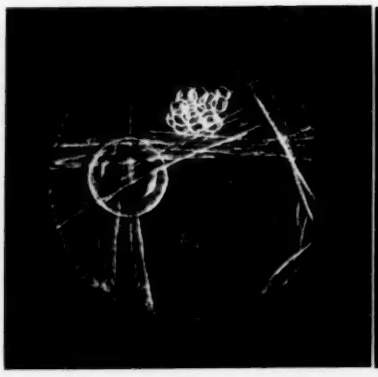

FIG. 5

FIG. 6

FIG. 5 — (Cowan and Fry) — Mrs. M., age 63. Drawing made 2 years after combined operation for senile cataract. When seen 3 months later the large globule had disappeared.

FIG. 6 — (Cowan and Fry) — A. B., age 11. Seen 8 days after unsuccessful discission for congenital dislocated cataract. The knife needle merely nicked the lens capsule, at which place the bunch of globules grew.

FIG. 7 — (Cowan and Fry) — Section through the secondary cataractous membrane, showing anterior lens capsule, and proliferated capsular epithelium.

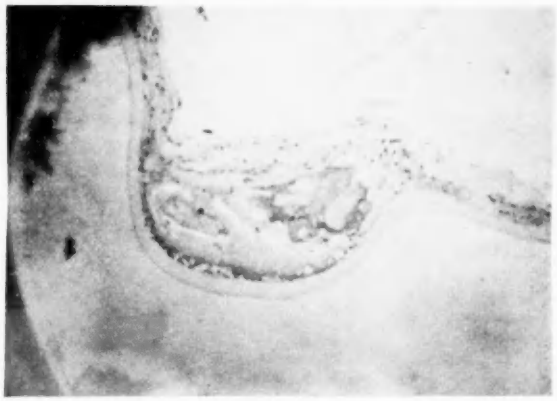

FIG. 8 — (Cowan and Fry) — Fold of the anterior lens capsule within which are seen large bladder cells and proliferated capsular epithelium with adjacent, interlacing hyaline bands.

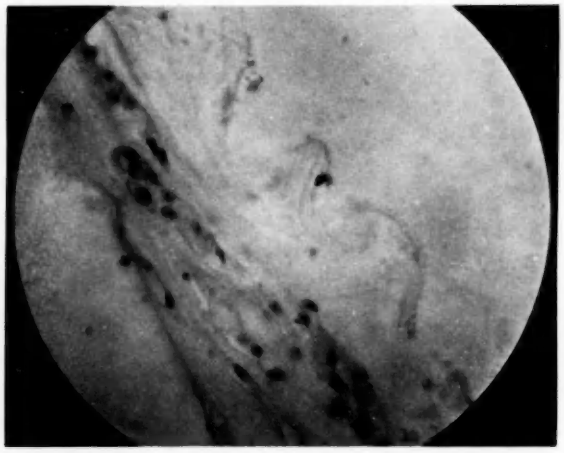

FIG. 9 — (Cowan and Fry) — Proliferated capsular epithelium with loops of apparently newly-formed capsule tissue.

FIG. 10 — (Cowan and Fry) — Capsular epithelium which has proliferated to cover both sides of the anterior capsule.

FIG. 11 — (Cowan and Fry) — Two vesicle-like capsular epithelial cells. They appear different from "bladder cells."

FIG. 12 — (Cowan and Fry) — Vacuoles within a lens dislocated into the vitreous. The lens capsule was intact.

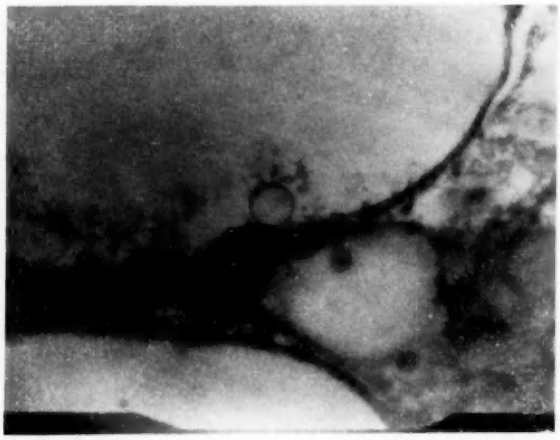

FIG. 13 — (Cowan and Fry) — Vesicle-like formations beneath the capsule of a lens dislocated into the vitreous.

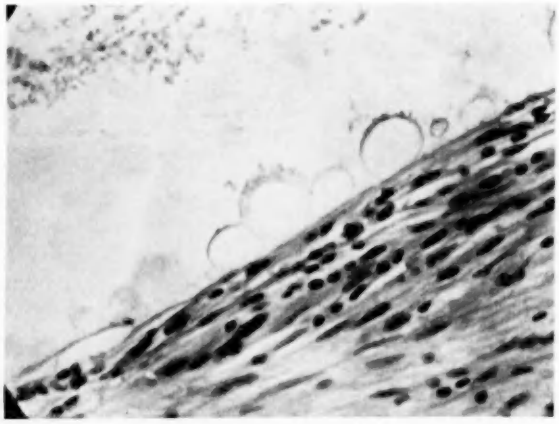

FIG. 14 — (Cowan and Fry) — Group of vesicles on the surface of a degenerated retina.

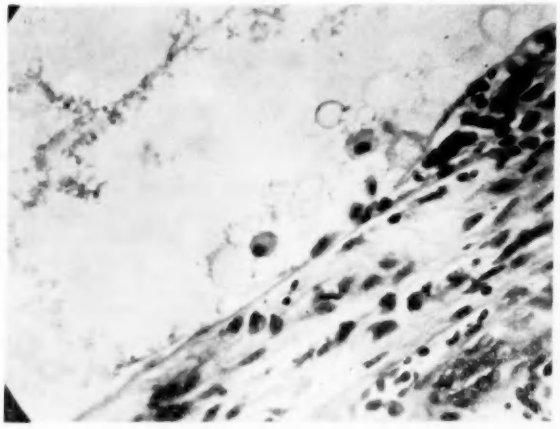

FIG. 15 — (Cowan and Fry) — Group of vesicles, apparently derived from cells on the surface of a degenerated retina.

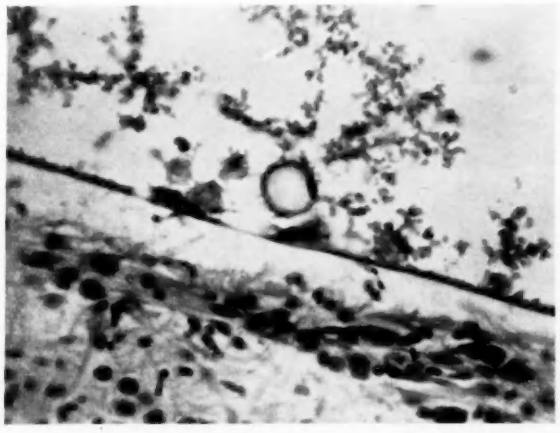

FIG. 16 — (Cowan and Fry) — Vesicle on the surface of a degenerated retina, in which a flattened nucleus is visible, indicating the cellular origin of the vesicle.

DISCUSSION

DR. HARRY S. GRADLE, Chicago: There is really very little to add to what has been said regarding the phenomenon of the so-called Elschnig bodies. That is not uncommon. We have all seen them in our cases. If we have not had them at all, we have simply referred to them as the presence of Elschnig bodies and not paid any particular attention.

As far as I know, there has been very little written upon the subject. In looking up the literature I was able to find some four odd references, all in the foreign literature. Interestingly enough, despite the careful observations made, this paper by Drs. Cowan and Fry is the first one that I know of published in English. Probably their paper will stimulate other investigators to note the occurrence of these bodies more particularly and possibly they can work out a clinical causation.

As far as adding to the paper, I have very little to say. I have never examined the case histologically. I have seen some of them clinically and I have attributed no significance to them whatsoever.

THE USE OF CONCENTRATED EPINEPHRIN PREPARATIONS IN GLAUCOMA, IRITIS, AND RELATED CONDITIONS

A CLINICAL STUDY*

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SAINT LOUIS

THE ACTION, effects, and clinical use of Epinephrin in various forms has been the subject of numerous contributions since Wessely first described its effective lowering of intraocular tension. The subject is well covered by the reports of many distinguished writers including Gifford,[1] Gradle,[2] C. Hamburger,[3] Thiel,[4] Post,[5] Howell,[6] and numerous others. Still it seems justifiable to report our experiences in the clinical use of this agent in a large number of cases of different types encountered in the course of several years' office practice. The study of these cases and of some of the excellent recent articles in the literature enables us to form and present some fairly definite ideas as to what one may expect in different kinds of cases.

One of the most important lessons we have learned is that any given case may respond in an entirely unexpected manner and that concentrated Epinephrin should first be used only where the patient can be kept under observation for an hour or more after its first application. If no ill effect is noted in this period, usually none will arise later.

The preparations we have used and which are suitable for office use are:

1. Epinephrin Hydrochloride, 1:1000 solution. This is used by subconjunctival injection. One-fifth to one-third cubic centimeter injected at the limbus is a powerful mydriatic and causes a marked lowering of intraocular pressure. The latter effect does not pass for forty-eight to seventy-two hours and may last even

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longer. This is true both of normal eyes and those with simple glaucoma.

The unpleasant systemic effects of palpitation of the heart, tremor, sweating, coldness of the extremities, rising blood pressure, weakness, vomiting, or even collapse are more probable with subconjunctival injections than with other methods of administration though they may follow in any case regardless of method employed.

Those suffering with toxic goitre, diabetes, sympathi-tonic individuals are most susceptible to these unpleasant effects. Our experience, as that of others, indicates that there is little actual danger to life and that repeated use of the remedy leads to lessening of these reactions.

Epinephrin Hydrochloride solution may be introduced into the conjunctival sac by well saturated cotton-tampons wound on a smooth applicator after the method of Gradle. The effect is somewhat less marked than after subconjunctival injection, but still wide mydriasis and marked lowering of tension follows.

2. Epinephrin Bitartrate is available in the American market in glass ampoules containing .091 gm. (.05 gm. active principle). This may be employed as:

a. Solution. One ampoule dissolved in 4.5 cc. distilled water gives the two per cent solution ordinarily referred to. Stronger or weaker solutions may be employed. This solution is rapidly discolored and is unfit for use after ten days or two weeks. Employed as drops in the conjunctival sac or by cotton tampons, it has powerful action as a mydriatic and in lowering tension.

b. Ointment of Epinephrin using equal parts of petrolatum and lanolin as a base in 4.5 gm. or 9 gm. tubes gives a two per cent or one per cent product that is stable and convenient to use.

The action following the instillation of the salve into the conjunctival sac is more gradual and less violent than after the use of similar concentrations in solution. The salve is retained longer in the sac; hence the effect is more prolonged. Systemic symptoms are not so liable to follow but are not unknown.

c. Epinephrin Bitartrate in Tragacanth jelly in concentration of one per cent or two per cent, dispensed in tubes, provides a stable, convenient means of application.

This preparation was described by one of us[7] at the meeting of this Academy in Chicago in 1934. This, as stated at that time, is a convenient and economical method of use both for office and prescription purposes. The rapidity of action seems to be between that of the solution and the ointment of like concentration. After using the jelly several weeks or months, some patients develop a conjunctival sensitiveness to it. The ointment may then be substituted for it.

3. Glaucosan (Links) as described by Hamburger is a two per cent solution of Lævo-Epinephrin with similar amount of one of the preliminary substances developed in the production of synthetic Epinephrin, Methylanimoacetobrenzcatechin.

This preparation is said to give the mydriatic and tension lowering effects of Epinephrin without the unpleasant systemic results. It is unstable and is to be used only immediately after opening the ampoule; thereby making its use a matter of very considerable expense.

Since beginning the use of the Epinephrin Bitartrate in jelly form, our employment of this agent has increased many times because of the convenience of having a stable preparation always at hand and being able to prescribe it economically for home use.

Chronic simple glaucoma is the disease in which many writers on the subject have found Epinephrin concentrate most useful.

Darier noted the tension lowering effect of Adrenal-extracts in glaucomatous eyes and recommended their use with miotics.

Rubert described the three-phase effect of Epinephrin on the ocular tension; first, a slight drop; second, a brief rise; third, in glaucoma, a drop lasting often several days.

MacCallan, Jessop, Senn early warned against unrestricted use on account of danger of acute rise of tension in some cases. Erdmann recommended Adrenalin as a means of prolonging and enhancing the effect of miotics.

Thiel, by careful clinical observation, determined the pressure curve after Epinephrin. His method was to measure the pressure at 7-8 a.m., then instil the salve three times at one hour intervals, at 8, 9, and 10 a.m., after which the tension was measured frequently for several days. He described a three-phase effect.

First phase. The pupil dilates in twenty to thirty minutes reaching its maximum size in two to three hours. The ocular pressure is generally unchanged, at times a slight fall, or perhaps an acute rise.

Second phase. From noon to early evening an abrupt fall of tension, lowest point may be reached the next morning.

Third phase. Pupil slowly contracts to its original size in seventy-two hours. Tension rises slowly. The effect varies as to duration from two or three days to as many weeks.

The explanation of these effects that seems most acceptable is as follows:

The early rise of tension is due to dilatation of the pupil.

First lowering of pressure is due to vasoconstriction, reducing the arterial supply and draining the previously engorged choroidal vessels.

The prolonged lowering of pressure is due to secondary paralysis of uveal tract vessels increasing venous drainage and quickening the blood flow.

Our experience with Epinephrin preparations in chronic simple glaucoma was not so encouraging as to lead to its adoption as a routine measure. Many cases in which it was tried seemed to show little or no response. Our practice was, therefore, to employ miotics for a period and to recommend early operation. Only in cases where operation was to be avoided for some special reason did we use Epinephrin in conjunction with miotics for prolonged periods. We found it useful in re-activating the eye to miotics after the latter had seemed to lose effect.

Case No. 1. Mrs. M. B., age 67. LaGrange operation in 1933 for chronic secondary glaucoma led to loss of vision in the right eye after a long course of treatment and repeated operations. The left remained unaffected until May 1934 when tension was 33,* vision normal. Under influence of Pilocarpine in increasing strengths, tension remained near normal level, frequently rising to 30 or 33 even while using Pilocarpine 4%. February 25, 1936, tension for 26; Epinephrin Bitartrate 2% salve was instilled; tension in one hour was 22. This salve was prescribed for home use twice daily with Pilocarpine 4% solution three times a day. Tension remained constantly 16 to 22 except when Epinephrin salve was omitted. April 14th, 1936, tension was 37 and Eserin 1% in castor oil was prescribed to be used with Epinephrin. Tension has been well controlled at level of 18 by alternating Pilocarpine solution with Eserin preparation and using Epineph-

* The numerals denoting tension in this article indicate millimeters of mercury as measured by the Schiötz tonometer.

rin twice daily up to September 10th, 1936. It is probable that this will cease to be effective and operation will be necessary.

The effectiveness of Epinephrin as an adjuvant to the miotics is illustrated in the following:

Case No. 2. E. J. D., age 30. October 10, 1930. Dull headaches; halos; tension 37; each eye, vision normal. Pilocarpine solution in increasing strength was used till November 7th, 1930, when tension was 33. Pilocarpine 2% solution instilled in each eye with no reduction of tension in one-half hour. Glaucosan (Links) drops were used and in one hour tension was 22 in each eye. Pilocarpine 1% solution used three times daily with tension remaining at this level till date of operation, two weeks later, when bilateral trephining was done. Tension and vision were normal four months later without treatment.

After operation for glaucoma there may be a rise of tension difficult to control by use of miotics. Such a case may respond to Epinephrin.

Case No. 3. W. L., age 55. Left eye blind; right eye, vision failing two years; field contracted; vision 5/7.5; disc, deep glaucomatous cup; tension 26. Trephining right eye, November 14, 1933. December 11, 1933, tension, right eye, 37.

Miotics used vigorously till August 20, 1934, with tension fluctuating, 22 to 37. Epinephrin Bitartrate 2% in jelly prescribed for use twice daily. Tension at once dropped to 26 and remained so till time last seen, January 28, 1936, under continued use of Epinephrin jelly twice daily.

Case No. 4. Mrs. A. L., age 47. Acute glaucoma, left eye, in 1929, relieved by miotics, but developed chronic glaucomatous state with tension fluctuating from 22 to 50 under treatment with Pilocarpine and Eserin until November 25th, 1935, when trephining was performed. December 5th, 1935, tension again rose and remained in range 26 to 30, under use of Eserin 1% in oil four times daily. Epinephrin 2% in jelly instilled. One-half hour later, tension 22. Prescribed for use once daily. Eserin discontinued, tension remained constantly at 18; all medication gradually withdrawn by March 5th, 1936. Last seen July 29th, 1936, tension normal.

Chronic simple glaucoma may be transformed into an acute fulminating attack by Epinephrin used one time. This fact is now well known and mentioned by many writers on the subject. This case impressed it upon us many years ago.

Case No. 5. Mrs. J. D., age 75. Suffered an attack of acute congestive glaucoma in right eye August 1926. Relieved by trephining and still remains quiet with normal tension. October 23rd, 1929, patient appeared with tension of 47 in the left eye without other signs. Vision 5/6. Glaucosan (Links) drops instilled. Before reaching home an acute congestive attack had developed; tension 60. Partially relieved by frequent use of miotics and heat. October 30th, 1929; tension 37. Trephining led to gradual quieting of the eye. The tension has remained normal to date. Vision now reduced to 5/30 by lens opacities.

Chronic secondary glaucoma with uveitis is a type of case in which Epinephrin concentrates are not recommended generally. Nevertheless we have had occasional good results and consider their use if miotics are not effective. If the tension rises after first treatment while the patient is kept in the office for observation, as he should be, paracentesis at the limbus followed by Eserin ointment serves to bring relief.

Case No. 6. Mrs. H. P., age 70. Seen October 28th, 1935, by Dr. W. E. Muldoon who noted episcleral redness, tension 20, cornea clear. November 18th, 1935, he reports lower third of cornea clouded in all layers, striation of Descemet's membrane, homogeneous deposit on post surface. Tension 30. Miotics had been used. December 3rd, 1935, we found corneal epithelium edematous with blebs and an ulcer. Tension 26. Paracentesis was done. December 6th, 1935, tension 30. Epinephrin Bitartrate 2% in jelly used in sac. One hour later tension 24. Prescribed for home use three times daily. Tension remained 18 to 22 till January 28th, 1936, and treatment was discontinued. No further rise has been noted.

In acute secondary glaucoma with uveitis, Epinephrin may be helpful or may lead to a painful rise in tension. It is our practice to attempt to reduce the tension by miotics and hot fomentations while treating the uveitis vigorously by constitutional measures, the most effective of which is the use of typhoid vaccine intravenously in non-specific cases. Dilating the pupil by Epinephrin serves to prevent adhesions and may reduce tension.

Case No. 7. Dr. A. C. H., age 48. First seen November 28th, 1931, with eye red and painful for two days; tension 55. Use of Eserin ½% and Pilocarpine 1% alternately every one and one-half hours reduced the tension to 37 in two days. The vision remained 5/6. Glaucosan (Links) drops dilated the pupil and in one hour the tension was 33. Epinephrin Chloride, 1/1000 solution was prescribed to be used by tampons at home. Next day there was much pain; tension 47. Eserin Alkaloid 1% in castor oil prescribed. This produced a fall of tension to 37, but in two days there was a rise to 47 and well-defined uveitis was present. La Grange operation for glaucoma was followed by satisfactory recovery.

Case No. 8, seen this summer, illustrates another valuable manner of using this drug.

R. G., age 52, was referred at his own suggestion because he wished to avoid an operation for glaucoma recommended by his local oculist. He had suffered a mild iritis for three weeks which had at first responded to local and general measures only to develop a painful rise of tension to 55 on the day he was referred. By evening, the use of Eserin and hot fomentations was followed by a drop in tension to 26. Their continued use at two hour intervals with a bacterial antigen intramuscularly quieted the eye in one week and the patient returned home with tension 18 and only a few cells in the aqueous. Almost complete recovery was interrupted four weeks later by pain, deep ciliary redness, very heavy pigmented adhesions of the contracted pupillary border to the lens. The tension remained low. The aqueous was filled with clumps of cells and showed a marked flare in the slit lamp beam.

Epinephrin Bitartrate 2% in jelly base caused severe pain but only slight dilation of pupil at one point in two hours. A second instillation with addition of Atropine powder was made and a dressing applied for two hours. Atropine Alkaloid 1% and Cocaine 1% in oil were prescribed for use every two hours.

The next day the pupil was still small; tension low; but a few more adhesions had yielded. Epinephrin solution 1/1000 and Atropine 1% was then injected subconjunctivally and the lower half of the pupil dilated to a width of four millimeters in two hours. Epinephrin jelly again instilled, and dressing applied for two hours. Atropine and Cocaine oil used every two hours at the

hotel. Next day the pupil was again small, but aqueous was much clearer. Adhesions were present across top of pupil. The patient was then sent to the hospital where the same line of local treatment was augmented by the intravenous use of typhoid vaccine. Each morning the pupil would be small until the Epinephrin jelly was used, although Atropine was used each two hours. By the fourth day all adhesions were broken and the pupil was round. Most of the redness and all the pain was gone. Only a few cells remained. It was felt that only the powerful dilating effect of the concentrated Epinephrin had enabled us to break up these very dense adhesions at the same time its influence in reducing tension was of value.

Our experience with Epinephrin in cases of acute glaucoma agrees with that reported in numerous articles by other observers. In the beginning we used it persistently in several cases, occasionally producing a drop in tension of a few points after an hour or two but causing intense pain and at other times increased pressure. A review of some of our records of early cases confirms the opinion that acute congestive glaucoma is a contraindication to the use of any form of Epinephrin.

Following needling of soft cataracts in young adults, the tension often rises as the lens substance pushes forward into the anterior chamber.

We have used Epinephrin in several such cases, as tampons, Glaucosan (Links) drops, and as Epinephrin Bitartrate 2% jelly, with gratifying results. We quote only one of our early experiences.

Case No. 9. Miss C. T., a teacher, age 31; had a needling of a congenital cataract followed in twenty-four hours by marked rise of tension with pain and redness. Epinephrin Chloride $\frac{1}{1000}$ was applied on well-soaked tampons several times daily. In forty-eight hours the tension was low and absorption proceeded without further complication.

One of the most difficult types of glaucoma to relieve is that following cataract extraction. This most often occurs after needling of secondary capsular membranes with herniation of vitreous into the aqueous chamber. It is well known that such cases often do not respond to operative measures nor to the use of miotics. Many of them go on to complete blindness in spite of all treatment. We have used Epinephrin Bitartrate 2% jelly in many of

these cases, with gratifying results. Practically none of them has shown a rise of tension even on first application. Several have reached a state of apparently permanent low tension without further use of medication. Some require continued use of miotics along with Epinephrin. A few illustrative cases are cited.

Case No. 10. A. E. H., age 40. Had a mature cataract of complicated type in the left eye in October 1931. A mild glaucoma developed and the cataract was extracted extracapsularly in May 1932. Needling of capsular membrane, July 1932, resulted in vision of 5/6 but tension ran a stubborn up and down curve under vigorous use of Eserin and Pilocarpine along with two operative procedures — iridencleisis September 1934 and May 1935. September 1935, tension ranged about 30 under regular use of Eserin, and the eye was red and irritable. Epinephrin Bitartrate 2% in jelly was followed by fall of tension to 22. It was used twice daily for one month with tension constantly 16 to 18 when treatment was stopped September 30, 1935. No further rise of tension occurred till January 4th, 1936, when, following a blow by a soft ball, the tension was found to be 43. Epinephrin 2% jelly again used twice daily was followed by tension of 22, where it remained without further treatment.

Case No. 11. J. L. R., age 69. Cataract extraction August 1935. One month later, eye quiet; vision 5/10. A month after this he suffered a small hemorrhage into vitreous, reducing vision to 5/60, tension 43. After several days' treatment with miotics, Epinephrin 2% jelly was instilled and prescribed for use twice daily. Two days later tension was 26. In one week, tension 18; vision 5/5; treatment stopped. Last seen one month later, condition unchanged.

Case No. 12. Mrs. J. M., age 39. Had leukoma of cornea and cataracts as result of reaction from eyelash dye. Cataract extraction March 1934; iridocapsulotomy September 4th, 1934. Next day, tension 37. Under use of miotics tension varied from 22 to 37 till April 18th, 1936, when it was 33. Epinephrin Bitartrate 2% jelly used once daily with Pilocarpine 1% twice daily; the tension became and remained normal, being 22 when last seen June 9th, 1936.

Case No. 13. Mrs. O. W. F., had a needling of an after-cataract capsular membrane January 10th, 1936. Tension rose

next day and remained up under use of Eserin and hot fomentations. January 13th, the tension was 43. Epinephrin Bitartrate 2% jelly was instilled. Next day, tension 37; with pain and blurred vision. Epinephrin again used. Some cardiac distress with rapid pulse and tremor. In one-half hour tension 26 and eye comfortable. January 15th, tension 30, had used only Eserin. Given Epinephrin jelly to use three times daily at home. January 17th, tension 18. This treatment was continued for two months, increasing the intervals, and then stopped. Tension remained the same on all visits and vision slowly cleared. Last seen two months after treatment was discontinued May 4th, 1936, tension 18; vision 5/6.

Numerous similar cases could be cited. Some respond after very brief treatment; some maintain normal tension by use of Epinephrin ointment alone; and others require both ointment and miotics. Some, of course, do not yield to any combination of measures and continue to blindness. In this group of cases, recognized as most difficult to handle, we feel that strong Epinephrin preparations, especially in water soluble ointment, are very effective.

The use of Suprarenin in cases of iritis with synechiæ as an adjuvant to Atropin in securing dilatation and freeing adhesions is well illustrated in Case No. 8, above. It has been so used in many of our private cases and by the staff of the Washington University Out-patient Department in a great number of staff cases. Its use is usually followed by considerable pain as the adhesions break away, but this can be controlled by sedatives. Acute rise of tension in this type of case is rare.

Summary

We have recorded here something of our experience in using strong Epinephrin preparations over a period of years, in many and varied cases. This experience and the rich literature on the subject have brought us to certain conclusions which we feel constitute a fairly comprehensive set of working rules for the use of this important therapeutic agent.

First: Concentrated Epinephrin is a powerful mydriatic capable of lowering ocular tension, or of producing an acute rise of tension, of causing severe pain, and occasionally unpleasant gen-

eral symptoms; hence, the first use should be made where the patient can be observed for one or more hours.

Second: Epinephrin is a valuable aid in chronic simple glaucoma where miotics must be used for prolonged periods in lieu of the preferred surgical measures.

Third: Chronic secondary glaucoma generally does not yield to Epinephrin but may do so, and a trial is justified.

Fourth: After operation for glaucoma, Epinephrin may be effective in reaching a state where the tension remains normal for long periods without treatment.

Fifth: Glaucoma, after needling of cataracts and after-cataract membranes has been controlled in a large percentage of cases by Epinephrin with or without miotics.

Sixth: Epinephrin is invaluable in breaking up adhesions in cases of iritis and uveitis.

Seventh: The most convenient form for office and prescription use is as an ointment in water soluble base (Tragacanth jelly) or in an oily base (Petrolatum and Lanolin).

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DISCUSSION

Dr. E. C. Ellett, Memphis, Tenn.: We have gone a long way in our knowledge and application of adrenalin since Darier first introduced it to ophthalmology in 1896 as an agent to dilate the pupil and reduce the intraocular tension. The present manner of employing it may be said to date

from 1922 when Carl Hamburger advocated the use of adrenalin or suprarenin in the treatment of simple glaucoma, later developing the drug called glaucosan. This is a combination of dextrorotatory suprarenin, or ordinary suprarenin, and methylaminoaceto-pyrocatechol, a polariscopically neutral by-product in the synthetic manufacture of suprarenin. This was intended for subconjunctival injection.

Continuing his researches, he developed a synthetic levorotatory tartaric acid alkaloid derived from the adrenal gland, which he called "Linksglaukosan" (levoglaucosan). This, used in two per cent solution and combined with methylaminoaceto-pyrocatechol in the same strength, proved to be active enough to use by instillation only. It causes a dilatation of the pupil and a decrease in intraocular tension. This is the preparation we have used and is what we mean by "glaucosan." It comes in sterile glass ampoules in two per cent solution and in the combination referred to, ready for use.

The method of instillation of levoglaucosan is simple. The eye is anesthetized with holocain, pantocain, or butyn, and then, with the patient in a recumbent position, two drops of the solution as furnished in the ampoules are instilled in the cul-de-sac. While the lids are held open, the patient is directed to rotate his globe in the four cardinal directions, which allows the drug to come into free contact with the globe. The drops are repeated from three to five times at intervals of fifteen minutes. In a few moments after the first drop, the eye should become porcelain white, the lid aperture widen, and the pupil dilate quite regularly, while a zone of ischemia usually develops on the lids, cheek, and temple. Practically always there is a distinct drop in tension. Sometimes an appearance develops on the cornea as if some glistening powder had been sprinkled on it. This soon passes off.

There has recently been available, thanks to the efforts of Dr. John Green, a synthetic epinephrin bitartrate, dispensed as a powder in glass ampoules. Each ampoule contains 0.091 grams of the drug, and when dissolved in 2.5 cc. of water, makes a 2 per cent solution. At the suggestion of Dr. Melville Black we have used this in the powdered form, taking up a small amount on a very small eye curette. The solution is not stable, but the powder is, and the use of the powder is satisfactory and economical.

As regards the use of these suprarenin preparations, we can speak favorably of their use in iritis, especially in those troublesome cases when the tension is increased. The pupil can be widely dilated, adhesions broken and the tension usually reduced, at least, never elevated. The dilation can often be maintained with atropin, but not always, and subsequent applications of the suprarenin seems much less effective than the first one. I have published my observations with many illustrative cases, to which I could add many more. The effect in breaking adhesions and fully dilating a rebellious pupil is very striking, and the pain, when present, is usually relieved.

Without apparent cause the application is sometimes very painful. We use it with the patient lying down, and precede it with ten grains of aspirin. Even so, a hypodermic of some opiate should be at hand. The unpleasant effect does not interfere with the dilation of the pupil, nor is it due to hypertension.

In glaucoma the results are not so good. Sometimes, but not always, the tension in simple glaucoma can be reduced, and can be subsequently kept under better control with myotics, but the result is not constant, and at best it is only an adjuvant to myotics, and sometimes it will precipitate a severe attack of acute glaucoma. The form of glaucosan which was introduced as especially effective in acute glaucoma, namely, amino-glaucosan, has proven a flat failure in my experience. It neither contracts the pupil nor lowers the tension.

The following cases might be mentioned as illustrating what the strong solutions of adrenalin can do in glaucoma simplex:

J. G., a man aged 60, had been under observation for nearly twenty years with slowly developing senile cataract. An intracapsular extraction was done on the right eye in November 1935. A small peripheral iridectomy was made and the eye healed without any complication except a temporary choroidal detachment. The fellow eye was operated in February 1936. The capsule tore and the lens was expressed, leaving some cortical matter which absorbed slowly. A month later the tension seemed to be elevated to the fingers, and on taking the tension, it was found to be 52 in each eye (Schiötz). Eserine promptly reduced it in the left eye to 18, where it remained, and on June 24 the eserine was stopped in the left eye and the tension remained 18. The right eye, while surgically perfect, was slower to yield, and the tension stayed at 30 to 40 till July 6, when adrenalin was applied. The pupil dilated and tension fell to 20 and the next day to 11. It subsequently rose to 37, but continued use of eserine has brought it to 22, where it remains for a time as long as the eserine is continued, but the adrenalin has to be repeated about every ten days. Vision is normal in both eyes with glasses.

Mrs. J., aged 32, was hyperopic 3.50 D, and in 1921 had an exudative choroiditis in O. D., from which she recovered with a large pigmented lesion above the macula, and vision slightly impaired. In March 1935, she had an acute attack of glaucoma in O. D., with rather typical prodromes, and vision was reduced to 5/200, tension 70. Treatment reduced tension to 32, but it was hard to keep it below 40 and the widely dilated pupil did not contract at all. A trephine with complete iridectomy was done, with relief of all subjective symptoms, but the pupil is still widely dilated after more than a year, and tension stays around 37 in spite of myotics. Adrenalin used several times has no effect on the tension. Vision is normal and field shows no change except what is accounted for by the choroidal scar.

Mrs. B., aged 50, developed a slight macular change in the previously healthy left eye in 1933. She had been under observation then for 13 years and the eyes were normal. A blood pressure of 186 mm. Hg. was the only thing to explain the eye trouble. The nerve gradually became pale and a little excavated, but not a glaucomatous cup. Vision is 6/6, field normal. In May 1936, tension was found to be 37 in this eye and 43 in the apparently normal fellow eye. The right eye has yielded somewhat to eserine, but tends to be 30 or more, the left eye yielding less and less readily. Adrenalin will reduce it in both eyes for a few days, but not below 26 mm. Hg.

NEVUS FLAMMEUS AND GLAUCOMA

K. L. Stoll, M.D.

CINCINNATI

It may be justifiable to report the following case of bilateral facial nevus flammeus with bilateral simple glaucoma, binasal hemianopia and other ocular, general and intracranial complications. The progress of some symptoms and the development of others could be followed during the observation of this case.

REPORT OF CASE

M. K., white, male, aged 23, presented himself in October 1934, stating that his vision had been failing for eighteen months. He had so-called infantile paralysis when six months old, could not keep his head up as an infant, learned to walk slowly and attended the school for crippled children from his eighth to sixteenth years of life. A so-called birth mark involving both sides of the face was noted at birth. The family history furnished no information.

GENERAL PHYSICAL EXAMINATION

No disorder of the inner organs was present, blood sugar, sugar tolerance, blood count and fragility test were normal. The number of the platelets was lowered, but their shape was normal. The nose, sense of smell, throat and hearing acuity were normal. Caloric irritation revealed marked hyperactivity of the right labyrinth. The metabolism was 30 points below the normal rate, obesity existed. The neurologic examination, done by Dr. H. Fabing and Dr. A. R. Vonderahe, showed hyperactivity of the deep reflexes on the right side of the body, then paresis and atrophy of the right arm and leg with marked motor weakness, Babinski's sign on the right foot and somewhat lowered mentality. Some minor disorders of the sympathetic nerve were present. The peripheral sympathetic nerve was considered uninvolved, half-sided facial atrophy was absent. No involvement of the peripheral trigeminal nerve was found, no sensory or motor disturbances existed.

FIG. 1 — (Stoll) — Photograph of a patient, showing the nevus of the face and the atrophic right hand.

DESCRIPTION OF THE NEVUS FLAMMEUS

The nevus flammeus involved the area of the first and second divisions of the trigeminal nerve of both sides of the face. The nevus was of a purplish hue, it was more of the dotted and streaked type on the right side of the face, and more uniform and thickened on the left side. The lids and left ala nasi were involved and the left portion of the upper lip was thickened. The left portion of the palate, uvula, gums and mucous lining of the left cheek showed arterial and venous hypervascularization producing a purplish discoloration.

OCULAR EXAMINATION

The function of the eyelids and extrinsic eye-muscles was normal. Nystagmus existed on forced look upward and to the left side. The conjunctiva of the eyelids was thickened by hypervascularization, but inflammatory symptoms were absent. The conjunctiva of the eyeballs had a purplish hue, it was thickened, semitransparent and of a gelatinous appearance, suggesting a

lymphangiomatous condition. Numerous fine aneurysms at the limbus were noted with the slit-lamp, and telangiectatic veins of an uneven caliber were observed. An especially large convolution was noted near the temporal portion of the equator of the right eye. The episcleral vessels were distended and tortuous. The corneæ had a normal curve and normal diameters. No folds or tears in Descemet's membrane were seen with the slit-lamp. The anterior chambers were slightly deepened and the chamber angles were free. Gonioscopic and cycloscopic examinations after Troncoso[1] and Vannas,[2] respectively, were not done.

The pupils were fairly equal, round and somewhat narrowed by a miotic ordered elsewhere. The structure of the irides was practically normal, atrophy or enlarged blood vessels were absent. The crypts were less distinctly outlined than normal, and the pigment seam of the pupillary margin was very narrow. The color of the irides was dark brown, corresponding in pigmentation to the color of the hair, with one exception which developed later on: A portion of both lower quadrants of the left iris presented a grayish appearance, suggesting hypochromia.

Examination of the fundus showed clear media and a darker red reflex than normal. The retinal veins and arteries were distended and tortuous; arteriovenous compressions were absent. The disks were pale and the lamina cribrosa appeared pale in the deep excavations measuring about five diopters. A wavy choroidal discoloration temporal from the left disk could be seen in the photogram but not with the ophthalmoscope. The fields of vision were considerably contracted, the left field having been limited to a narrow temporal sector and the right to the fixation point.

The intra-ocular pressure measured from 35 to 40 mm. (new Schiötz) in the right eye, and from 45 to 50 mm. in the left. The vision amounted in the right eye to 15/19 with $+0.5 \bigcirc +0.5$ cax 180, and the left to 15/24 with $+0.5 \bigcirc +1.5$ cax 180. Comparison with former correction showed that the hyperopia had decreased steadily since December of 1924, when it amounted to 2 convex diopters in the right eye, and to 4.5 in the left.

ROENTGENOGRAMS OF THE SKULL

Enormous enlargement of the frontal sinuses existed, and all other pneumatic cells, including the mastoid cells, were greatly enlarged. No evidence of bone destruction was noted anywhere in the skull. The left antrum of Highmore was definitely opaque.

FIG. 2 — (Stoll) — Roentgenogram showing the shadow of the lipiodol in the left antrum and the enormously enlarged frontal sinuses.

Lipiodol formed a triangular opacity with wavy outlines and filling spaces all around it. Tortuous and calcified blood vessels were evident well within the skull over the occipital lobe, and the grooves of the meningeal arteries were very distinct, some of them with double outlines.

THERAPY AND COURSE

Miotics succeeded in regaining almost full vision with the same correction of glasses, and in lowering the tension temporarily. The field of vision in the left eye increased, while that in the right eye contracted to the fixation point.

Iridotasis was done in both eyes, two weeks apart, in May of 1935, resulting in preservation of normal vision and tension ever since that time. The following changes of the fields of vision took place: binasal hemianopia became manifest since June of 1936. The field of the left eye continued its pre-operative recovery. The field of the right eye remained contracted to the fixation point until June of this year, 1936, when it regained a narrow temporal area. The hypochromia in the left iris spread recently into the upper nasal quadrant. The lymph-angiomatous condition disappeared in the conjunctivæ, the distention of its blood vessels receded and the convolution near the equator of the right eye became decidedly smaller since the operation.

SIMILAR CASES REPORTED

There seem to be only four cases on record of bilateral facial nevus associated with bilateral simple glaucoma. They were reported by Beltman,[3] A. Knapp,[4] Dunphy,[5] and Biro.[6] Bilateral facial nevus with congenital bilateral hydrophthalmos were reported by Marchesani[7] and Perera.[8]

OPERATIONS REPORTED

Two authors, A. Knapp and Dunphy, reported on bilateral operations performed for simple chronic glaucoma associated with bilateral facial nevus.

FEATURES OF THE CASE

1. The right-sided hemiplegia may be considered congenital because it occurred and was associated with hemiatrophy prior to puberty. It was caused by the neuropathology in the left cerebrum, as demonstrated by the presence of calcified vessels.

2. That the right hemisphere was also involved is evidenced by the positive Babinski's sign in the right foot.

3. That the cerebral lesion occurred within the first three months after birth, or that it was probably congenital, is suggested by the fact that the patient could not hold his head up in infancy.

4. The following considerations point to the presence of a hemangioma in the left antrum of Highmore which was collateral with the more extensive development of the facial nevus and with the intracranial angiomatosis: (a) The shadow of the lipiodol differed from shadows indicating polypi. Multiple shadows have never been observed in the presence of polypi after injection of lipiodol into the antrum, according to the statement of the roentgenologist. (b) Polypi would have made their appearance in the nasal cavity sooner or later and they would have produced symptoms of congestion in it. (c) The patient never suffered from any nasal disturbances and "heavy yellowish" discharge was never noticed. Therefore, the presence of a pyogenic membrane is unlikely. (d) The fluid used for flushing the antrum reappeared completely clear and free from any shreds of tissue or mucous flakes. (e) It is reasonable to assume that the emissaries of the internal maxillary vessels to the lining membrane of the antrum were equally as supernumerous and distended as were the vessels of the left side of the face, mouth and cranium.

(f) Angiomatous formations in the antrum have been observed by several authors. Reamer[9] reported on a hemangioma of the face associated with an angioma of the antrum of Highmore.

GLAUCOMA

Conditions point strongly toward the vascular theory of "plethora" advanced by Beltman,[3] Hudelo,[10] Waardenberg,[11] and others. There existed ocular and circumocular hypervascularity, also distention of the retinal vessels and probably hypervascularization of the choroid judging by the dark red hue of the fundus. Intraocular angiomatosis was recently described by Prevec,[12] and Ludwig.[13] The important rôle played by the vorticous veins was pointed out again recently by Schieck[14] and Sondermann.[15]

CONCLUSIONS

1. This case seems to indicate that the facial nevus and allied angiomatoses were congenital, and that they originated in early fetal life.
2. Cerebral angiomatous disorders caused, soon after birth, paresis of the right arm and leg with secondary atrophy.
3. Hypochromia developed and spread in one eye during observation. Lesion of the sympathetic nerve may have been contributory to the pathogenesis of the glaucoma, but it cannot have been an etiologic factor in this patient.
4. The extensive hemangiomatous malformation of the face included the orbits and eyeballs. It caused insufficiency and inactivity of the circulation. Glaucoma followed as a consequence of these two circulatory disorders. They produced hypervascularity (plethora) within the eyeballs and the vicious circle of irregularities of the circulation of the blood and lymph.

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DISCUSSION

Dr. Arnold Knapp, New York: I cannot add anything to Dr. Stoll's interesting report of his unusual case and his complete survey of the literature other than to give the subsequent history of the case of bilateral glaucoma and nævus which I have been observing since 1926, and which was reported in the Archives of Ophthalmology in 1928.

It was a boy of 12 and a trephining was done on both eyes in December 1926 with a satisfactory result. There was no change until the summer of 1929, when, after an injury to the right eye in diving, a rupture of the conjunctival bleb took place followed by an intraocular inflammation and loss of sight in the right eye.

In 1930 the filtration in the left eye having gradually come to a standstill through contraction of the filtration scar, another trephining was done. This has succeeded in keeping down the tension to the present date, and today vision and field are holding their own.

The curious changes in the iris which made the case unusual, namely, the presence of pale grayish tubercle-like nodules, which on microscopic examination proved to be angiomata, have not changed in any way.

Dr. Ernest F. Krug, New York: Facial nevi with cerebral complications are not infrequent. The eye often escapes. The clinical picture is known as a neuro-cutaneous syndrome. Where we have the eye as a part of the picture we may have unilateral buphthalmos, congenital glaucoma in the infant with jacksonian seizures, hemiplegia resulting from hemorrhage.

(Dr. Krug put slides on the screen during his presentation, in illustration of his talk, but not supplemental to it.)

I am showing these sections of an eye in a case of facial nevus because in this eye there was no tension, simply because the anterior segment escaped in the malformation. The patient was first seen by me in 1931 because of loss of vision in the right eye and ophthalmos. The mother stated that at the time of birth the eye was imperfect and that the child had a peculiar mottling of the skin on the right side of the face. In October 1925 he had an attack of what was diagnosed as epidemic encephalitis. He slept for six weeks continuously and was delirious for another month. Upper and lower extremities became helpless and speech was impaired.

The young man was 21 years old when first seen by me for Dr. Samuel Brock of the Neurological Institute.

Examination: Ophthalmologic examination revealed the right eyeball proptosed slightly forward, downward and outward, and its movements were limited inward and outward suggesting retrobulbar extension of the process. The eyelids were thickened and the conjunctival vessels were full. The right pupil was semidilated and fixed and consensual pupillary reaction was absent, showing a definite involvement of the optic nerve. The media were clear except for a small macula at the lower limbus at 7 o'clock, which was most probably the result of trauma from instrumentation at birth. Ophthalmoscopic examination of the right fundus presented an unusual picture. The veins were so greatly engorged and tortuous that the disk was almost completely obscured. Where visible, there was no elevation of the disk. The vessels showed much thickening indicative of a prolonged progressive process. Where distinguishable, the arteries were not enlarged, and at no point was there any visible communication between the vessels. There was no pulsation of the vessels. Some migration of pigment along the vessels was evident.

The veins of the left fundus were full, but not tortuous. With correction —1.00 sphere —0.75 cyl. axis 180, the vision of the left eye was normal (6/6 and Jaeger 1), and the field was normal for form and colors.

On March 18, 1932, the patient committed suicide by strangulation. The right eye, orbital contents and the brain were removed on March 20. The pathologic findings of the brain will be reported in Brock's and Dyke's paper, previously cited.

At autopsy, an angiomatous lesion was found extending from the right retina along the right optic nerve, chiasm and tract to the right of the mid-brain and via the right superior cerebellar peduncle to the right of the cerebellum (dentate nucleus) which explained the entire symptomatology.

Gross examination of the brain and coverings showed the right cavernous sinus to be the site of an angiomatous lesion which became confluent with a matted, reddish, flat, angiomatous mass covering the right part of the optic chiasm. The optic nerve was considerably enlarged by a covering of angiomatous tissue.

On sectioning the brain, a bilateral internal hydrocephalus was disclosed, the left lateral ventricle being somewhat larger than the right. The third ventricle was also considerably enlarged. The angioma was seen obscuring the outlines of the right half of the optic chiasm and tract. It extended backward and involved the dorsal and medial part of the left tegmentum and the tegmental portion of the right mid-brain, replacing the right quadrigeminal bodies, the brachium conjunctivum and the right red nucleus, and encroaching upon the aqueduct. Passing along the right superior cerebellar peduncle (brachium conjunctivum) the growth entered the right cerebellum destroying most of the right dentate nucleus. In this location a rather large blood clot was found.

The gross autopsy findings were of the greatest interest in that they brought to light an angiomatous lesion extending from the right retina and right optic nerve, chiasm, and tract to the right mid-brain and via the right superior cerebellar peduncle to the right half of the cerebellum (dentate nucleus).

Sections of the pons and the thalamic region, stained by eosin, methylene blue, phosphotungstic acid, Mallory's connective tissue, Weigert's elastic tissue, Van Gieson, and Laidlow stains, disclosed the following:

"The tumor is composed of many thin-walled blood vessels between which is nervous tissue showing varying degrees of gliosis. This gliosis is for the most part quite moderate. Most of the neural elements seem well preserved. The intima of the blood vessels is composed of a single layer of flat cells. In most of the vessels there is only a thin layer of collagenous connective tissue beneath this, in which a few scattered smooth muscle fibres are to be seen in the phosphotungstic acid stain. No internal elastic membrane is present. In some of the larger vessels the media is distinct but quite narrow, only a comparatively few circularly arranged muscle fibres being present. No degenerative changes are found in any of the vessel walls."

DR. EDWARD B. DUNPHY, Boston: The frequent occurrence of angioma of the choroid in association with nevus flammeus of the eyelids suggests a common origin for these tumors. It is believed that growth of blood vessels is regulated by the development of the nerves as nevi of the skin usually show an arrangement corresponding to the peripheral distribution of one or more nerves. During the developmental period of the vascular system, some defect of the nerves might impair their regulatory influence and allow an abnormal growth of blood vessels to take place, thus forming a nevus.

Many theories have been propounded to explain the origin of the glaucoma in such cases. In most histological specimens, blocking of the filtration angle by angiomatous changes in the wall has been shown to exist. In other specimens, however, the filtration angle has been free and some other explanation must be sought. I am inclined to agree with the essayist that in some cases an increased formation of aqueous may be a factor in bringing about increased intraocular pressure by overburdening the filtration angle.

In all the cases examined histologically the intraocular capillaries have been shown to be dilated. We know that dilatation of the capillaries increases their permeability and causes an increased protein content of the aqueous. This increased protein content tends to produce an increased formation of aqueous by reducing the difference in osmotic pressure between blood and aqueous.

In a recent issue of the French Archives of Ophthalmology, Appelmans uses the term "encephalo trigeminal angiomatosis" or "nervocutaneous angiomatosis" to include cases of nevus flammeus, intraocular angiomas and intracranial angiomas. He feels that cases of von Hipple-Lindau disease can be included in this group. The histological characteristics of the retinal tumors in this condition are those of a capillary angioma.

A further correlation has been made by van der Hoeve in associating with Lindau's disease two other rarer diseases affecting the eye and nervous system in a somewhat similar manner, namely, Bourneville's disease and von Recklinghausen's disease. These are similar anomalies, each having a special predilection for a germinal layer, Bourneville's being ectodermal, Lindau's mesodermal, and von Recklinghausen's made up of each.

It is possible, therefore, to correlate a number of different clinical entities having in common a neurovascular origin.

Dr. F. Bruce Fralick, Ann Arbor, Mich.: L. W., male, age 26. Patient admitted to University Hospital June 1, 1936, with a history of blindness in the left eye for 5 to 6 years. This eye had never had good vision. It had diverged since being blind. About 10 months ago, dimness of vision was noted in the right eye, with a blind spot before the eye. There had been no pain in either eye at any time. The occurrence of haloes about lights was indefinite. His local physician diagnosed glaucoma simplex and prescribed pilocarpine once daily, but its use was not regular.

The family history was negative. There were nine children in the family and no other similarly afflicted individual.

Examination: Vision in the right eye was 6/30 and in the left eye was nil. With a $+0.75\,S\,+0.25\,C\times 102$ the R.V. was improved to 6/12. Consensual reflexes absent O.U. Direct and accommodative reflexes present O.D., absent O.S. The whole face showed a deep wine colored nevus extending from 2-3 cm. above the brows to the chin and laterally to the ears and into the hair line at the temples. The mucous membrane of the mouth was also involved as well as the anterior part of the palate. The left eye diverged 15°. Extraocular movements were normal. The conjunctiva of the lids and globes O.U. appeared injected, but on closer examination it was found that the vessels were very dilated and tortuous and were a part of the nevus picture. The corneas were normal and measured 13 mm. in the horizontal diameter. The anterior chambers were deep. The iris of each eye was normal to slit lamp examination. Pupils were round and regular in outline but dilated to 5 mm. There was no slit lamp evidence of an anterior uveal disease.

Fundus Examination: O.D. showed the media to be clear. The disc presented a large, deep glaucomatous cupping with very pale appearance. No retinal lesion aside from the vessels which involved mainly the veins. The veins were tortuous and anomalous, there being communications between many of the branches. No hemangiomatous involvement of the choroidal vessels could be seen but the fundus was rather deeply pigmented and thus might have obscured such changes. O.S. was identical with O.D. except that the glaucomatous cupping of the disc was more extensive and the disc was completely atrophic. Intraocular tension was 35 O.D. and 41 O.S., Schiötz. The visual field of the right eye was normal for form and slightly constricted for colors for 5 mm. target. Bjerrum scotoma was present.

Diagnosis: Nevus flammeus with glaucoma simplex.

General physical examination was negative.

Course:

1. Two per cent pilocarpine O.U. once daily reduced the tension to 27 O.D., 35 O.S., but at times the tension would rise as high as 42 O.D., 48 O.S., and trephine operation O.D. was advised.

2. Elliott trephine operation O.D., which was uncomplicated except that after the chamber was emptied it was noted that the globe did not develop hypotony but seemed rather to become even more firm. No pain was experienced by the patient but no fundus reflex could be obtained. Two days later the fundus examination revealed that a complete retinal detachment had developed and that the retina was lying against the posterior surface of the lens. Convalescence was uneventful as regards the

trephine operation and the height of the detachment gradually became less as the weeks passed. A single retinal tear was found in the periphery along the 10 o'clock meridian. When the eye had become quiet a diathermy operation for the retinal detachment was performed one month after the trephine operation. The diathermy operation was unsuccessful, as was a subsequent operation. The retina remains completely detached and the patient's vision remains at 1/60.

This case is presented merely to point out the possibility of complication which may follow operative intervention, this complication possibly being due to the hemangiomatic state of the retina and choroid.

DR. K. L. STOLL, closing: I thank Dr. Knapp and the other gentlemen for their discussion. Dr. Krug's case with its absence of choroidal changes and glaucoma would indirectly support my contention that choroidal plethora is one of the etiologic factors for the occurrence of glaucoma in these cases. Dr. Tyson (A. of O., 8:365, (April) 1932, and 13:491, (March) 1935) considers the condition of the uveal vessels as the salient point for the development of glaucoma in these cases; this view may be safely extended to the vorticous veins in support of the theory of intraocular plethora as a source of glaucoma. Dr. Krug's case and that reported by Dehogue (Ann. d'Oc., 167:580, 1930) are proof for direct continuity of hemangiomatosis of the face, eye and cerebrum.

Dr. Dunphy's reference to choroidal hemangioma is supported by Ludwig's compilation (Klin. Monatsbl. f. Augenh., 95:168, (Aug.) 1935). He refers to the incidental finding of choroidal angiomas in five eyeballs enucleated after the diagnosis or suspicion of sarcoma, and in thirty-eight eyes enucleated for glaucoma. Jahnke (Ztschr. f. Augenh., 72:354, 1930) enucleated an eye in a patient with facial nevus flammeus for absolute glaucoma. He found a flat hemangioma of the choroid which Jahnke considered the primary cause of the glaucoma. With reference to the cerebral types mentioned by Dunphy, I refer to Appelmans (Arch. d'Opht., 52:835, (Dec.) 1935), who stated that a clear division is impossible between facial angiomas with glaucoma and encephalo-trigeminal angiomatosis which is rarely combined with glaucoma. Appelmans and Schiötz (Acta Psychiatr. & Neurol., 10:Fasc. 4, 1935) referred to connections between nevus flammeus, epilepsy and the diseases of Bourneville, von Hippel-Lindau and von Recklinghausen.

Dr. Fralick's facial vascular nevus, I understood, was of a dark wine color. It would be interesting to know if it was one of the type which actually deserves the term of nevus according to Rintelen's view. Rintelen suggests the use of the term nevus only in those cases of nevus flammeus in which nevus cells were found (Klin. Monatsbl. f. Augenh., 94:463, (Apr.) 1935; abstr., A. of O., 16:531, (Sept.) 1936). I should prefer an iridotasis to trephining in eyes which present as extensive a vascularization as existed in my case.

SCLEROMALACIA — REPORT OF A CASE

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THE SCLERA consists of a dense, firmly-knit capsule of inter-woven meridional and equatorial fibres of connective tissue. Its vascular and lymphatic supplies are extremely limited. These factors, together with the sparseness of nerve terminals, both render the structure ideally protected against the invasion of micro-organisms and leave it peculiarly defenseless to repel infection or to combat influences making for degeneration.

The pathology of the sclera is briefly told. When scleritis, episcleritis, staphyloma and penetrating injuries, together with certain cysts and tumors, have been considered, the story is nearly complete. Parsons[4] added the more rarely encountered tubercle, leprosy, annular scleritis, and fatty and hyaline degeneration.

Scleral therapeutics are even more quickly disposed of. Hazy references to rheumatism, gout, lues and gonorrhea give but slight encouragement in a search for etiological factors in diseases of this structure. Thus the therapeutics and the pathology of the sclera provide one of the shortest chapters in textbooks on ophthalmology.

Rather recently van der Hoeve[6] reported several cases of scleral disease under the title of Scleromalacia Perforans which fall under a classification Rochat[5] previously termed Scleritis Necroticans. Van der Hoeve made the suggestion that these cases may be more frequent than is commonly suspected, and emphasized their probably definite connection with the severe polyarthritis present in the same patients.

The following case, to be briefly described, is one of interest as further emphasizing this connection. It is unusual in that it falls into none of the known classifications of scleral diseases and varies in certain particulars from van der Hoeve's perforating cases. Nothing resembling it can be discovered in literature nor

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has any similar case been encountered in the experience of several distinguished confrères who have examined the patient.

Since it is evidently a case of degeneration of the sclera, the name scleromalacia (following Professor van der Hoeve's nomenclature) seems most appropriate.

This patient, a woman, is now sixty-four years old. She has been a victim of rheumatism since the age of twenty-one. She took to her bed nine years ago by reason of a fractured hip and has been there continuously since because of a deforming polyarthritis. Practically all joints are affected. Her hands are gnarled, her feet less so. Her spine is nearly rigid. There is some movement of the knees, elbows and shoulders, and she can stand and even take a few steps if aided. Tophi are present in several joints.

My acquaintance with her began seven years ago when I was called in to pass upon the advisability of enucleating one eye, as counselled by a general practitioner.

At that time both eyes showed similar changes, more marked in the left. The first glance seemed to reveal a huge anterior staphyloma, though a moment's examination showed the fallacy of this observation as all bulbar measurements were normal. The unusual appearance was due to the color of the sclera, which had lost its normal whiteness and looked slatey gray. At a distance the sclera resembled the cornea with gray iris behind it. The conjunctiva appeared intact. The episcleral tissue throughout, after a stage of œdematous swelling, has, with gradually receding margin, been completely absorbed.

"Vitrification" of the sclera was the term that came to my mind at that time and that perhaps best describes an eye whose thinning walls are translucent, showing the choroid through. This vitrification has gradually extended further back, more rapidly in the superior than in the inferior quadrants, until today the change involves each eye, at least to the equator — how much further there is no means of determining.

When seen first the cornea and the other media were clear and permitted satisfactory intra-ocular examination. Such examination revealed no abnormality of the fundus. Tension has never been elevated. As the years have passed other signs of degeneration have occurred — both lenses are now cataractous and both eyes, except for light perception, are sightless. The left cornea has in recent years shown generalized clouding and roughening but without ulceration. I suspect that a uveitis has existed but it has been so insidious as to cause no symptoms. A rather recent devel-

opment is the occurrence in each iris of multiple radiating perforations, two or three in line, each perforation one to three millimeters by one millimeter, such radiating lines separated by distances of three or four millimeters around each iris. These perforations appear to be ruptures through the thin, atrophic iris.

At no time has pain been an important feature of the case. Such discomfort as the patient has suffered was described rather as "burning" and "irritation." As to the inception of eye symptoms, her recollection is that her trouble began with the common sensation of a foreign body in the eye. There is no history of trauma or of previous iridocyclitis. The palpebral conjunctiva appears normal, as do the lids and other adnexa. Bulbar movements are normal. Thorough general physical examination by a competent internist revealed no systemic disease apart from the chronic arthritis. Questions as to her customary diet suggested no vitamin deficiency.

In van der Hoeve's cases there were multiple perforations through the sclera at or above the limbus, in approximately the "12 o'clock" position, sometimes under an intact conjunctiva, sometimes opening through the conjunctiva. Careful examination with the loupe in this case reveals no area at which the sclera threatens to perforate. Observation under the slit lamp is impossible.

In the following particulars this case differs from those of van der Hoeve:

1. There is no perforation or evidence of threatening perforation of the sclera.
2. The episcleral tissue appears to have been absorbed, leaving no vestige of a limbus.
3. The multiple perforations of the iris are marked.

It is significant that all cases of this type involving degeneration of the sclera have occurred in patients suffering from arthritis deformans. A survey of the literature of this latter subject reveals the density of our ignorance as to its real nature and its cause. It is suggested that defective metabolism in the fibrous tissue and insufficient elimination of waste is a possible basis of arthritis deformans. This may be a factor in Scleromalacia. Friedenwald[1] stated that arthritis deformans is now regarded as a chronic infection. He, with a number of other writers, quoted Osler[2]: "Arth-

ritis Deformans is regarded as a chronic infection caused by certain varieties of streptococci and secondary to a focus of infection somewhere."

The American Encyclopedia of Ophthalmology[3] says "Inflammation of the joints is properly associated with ocular changes" but makes no more definite statement and does not mention scleral involvement. Eber[7] of St. Louis in 1934 reported under the term "Fistula at Limbus" a case of conjunctival bleb near the limbus and of over one year's duration, with a small opening through the sclera through which aqueous exuded. This patient had had rheumatic arthritis since childhood.

A recent exhaustive study of the entire field of arthritis entitled "The Present Status of Rheumatism and Arthritis with review of the literature"[8] makes no reference to ocular conditions or to complications involving the eye. Knapp[10] in his "Medical Ophthalmology" as well as certain other more recent works[12] make no mention of arthritis deformans.

Friedenwald[11] said "The one thing that can be said with certainty about the etiology of these intra-ocular inflammatory troubles is that it must be in some way related to the etiology of certain forms of arthritis for the association of these two conditions is far too frequent to be merely accidental."

One is therefore forced to the conclusion that we still have far to go in the study of the relationship between diseases of the sclera and severe joint inflammation. We must get what encouragement we can from such general statements as that of Adamson[9] that "The same poison may act on the individuality of tissue in different ways; just as champagne or port do, which in the one do no apparent harm, in another cause gout, in another headache, and so on. Thus might we expect the streptococci on a soil previously prepared by intestinal intoxication to produce rheumatoid arthritis in one, osteo-arthritis in another, or fibrositis in a third, and we can conceive that an antecedent focus of lesser resistance may determine an attack on different joints, one or many, the difference not being in the bacterial invasion, but in the physico-chemical state — can I say the electro-chemical state? — of the tissues." It is evident that the physiology of the joints merits much further study.

It is with the desire of making a contribution, however slight, to the solution of this problem of possible relationship between scleritis and arthritis that this report is offered.

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DISCUSSION

Dr. William L. Benedict, Rochester, Minn.: Dr. Kiehle took me out into the country at a recent visit to Portland to see this patient. He told me that other of his friends had seen the patient and none of them had ever seen anything like it. I never had seen anything like it. He had no reason for calling upon anyone else to discuss this paper and asked me to make some remarks.

I saw the patient in April of this year with a condition about as it was depicted upon the screen. It seemed to me that the sclera was not only undergoing a gradual process of absorption but that in certain areas there was an infiltration which could not be looked upon as inflammatory but probably a condition that we frequently see in malacia where there is a deposit of fat and other abnormal tissues in the region.

Furthermore, through the very thin sclera and through the nearly transparent conjunctiva appeared the atrophic uveal tract. The slate color of the eyeball evidently was due to the pigment in the uveal tissue showing through. The mottled appearance was due to the infiltration of the region which once had been occupied by the sclera.

It was rather striking to note the very marked changes in the sclera, with such insignificant change in the cornea. For many years after the scleral degeneration had been progressive the cornea had remained nearly clear even in its marginal portions.

We well recognize the changes that occur in the choroid in cases of inflammatory diseases of the sclera, in scleritis and episcleritis. I attribute the change of the choroid, the spaces in the iris and the opacity in the vitreous as secondary to the disease in the choroid and not necessarily a part of the process of which the sclera was the most prominent tissue affected.

The relationship between the disease of the eye and the arthritis, or fibrositis, is more than incidental. Evidently the inflammatory processes had gone much further in the joint and in the muscles and evidently some changes in the bones had taken place. I can only agree with Dr. Kiehle that both the changes in the eye and those of bones must be secondary to some certain primary cause.

DR. FREDERICK A. KIEHLE, closing: All I can add is to say that I am very grateful to Dr. Benedict for having gone out to see this patient with me. I had hoped that Dr. Weeks would join in the discussion. He has seen her several times and has spent hours in examining her. Col. Kirwain, of Calcutta, dropped in for a visit two or three years ago. I immediately bundled him into the car and took him out to see this case in the hope that it might suggest something that he had seen in the tropics. But, apart from agreeing with us that it was an absorption rather than a malignant ulceration, there was no further light thrown upon it.

In conclusion, I will merely say that I have a letter from Professor van der Hoeve in which he says that one of his cases showed a process of scleral absorption for some forty years before the sclera perforated, so that this still may fall into the classification of perforating scleritis.

Instructional Subjects

The American Academy
of Ophthalmology and Otolaryngology

Forty-First Annual Meeting
New York • 1936

LYMPHATIC DRAINAGE OF THE HEAD

* * *

LYMPHATIC DRAINAGE OF HEAD AND NECK AS SHOWN BY INJECTIONS OF INFANT AND ADULT HUMAN MATERIAL

WITH CLINICAL COMMENTS

OSCAR V. BATSON, M.A., M.D.

PHILADELPHIA

IF THE TIME permitted, it would be interesting to trace the historical development of our concept of the lymphatic system. We would find ideas recurring in cycles. The belief of one generation, considered adequately demonstrated by the methods of the times, after being denied by a succeeding generation, is later reaffirmed. In looking back we can pick out here and there statements which may be taken as almost adequate to express our present ideas. For example, William Hunter[1] said that, "I think I have proved that the lymphatic vessels are the absorbing vessels all over the body; that they are the same as the lacteals; and that these altogether, with the thoracic duct, constitute one great and general system, dispersed through the whole body for absorption; that this system *only* does absorb, and not the veins; that it serves to take up, and convey, whatever is to make, or to be mixed with the blood, from the skin, from the intestinal canal, and from all the internal cavities or surfaces whatever." Although Hunter was quite sure of himself, it is only now that this idea is reasonably well established according to present day physiological criteria. We are still in doubt about "all the internal cavities." Although the anatomists have located the major collections of lymphatic vessels and carefully mapped out the collection of lymph nodes, we must look to the physiologists for our knowledge of the rôle of the lymphatic system in the fluid absorption of the body. In the present discussion an effort will be made to state in general what are some of the accepted facts about the lymphatics of the head

and neck and to indicate where there are gaps in our knowledge, and to show from some first-hand observations some points which may properly interest the otolaryngologist.

In a normal economy, the lymphatic system seems, as indicated by Hunter, to be concerned with the return to the blood vascular system of fluid and protein which may have escaped from the blood. Alterations of this tissue fluid, by changes in the permeability of the capillaries in disease, leads to an increased burden on the lymphatic system and makes imperative a knowledge of its pathways. Moreover, the lymphatic system is capable of carrying not only fluids but particles, such as organisms. Certain malignant invasions are definitely known to follow lymphatic channels. There-

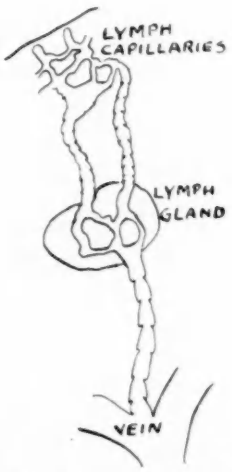

FIG. 1 — (Batson) — Schema of the lymphatic system. The numerous valves in the system are indicated by the constrictions.

fore, for diagnosis, prognosis, and for operative interference the location of the lymph ducts and glands must be known. One may visualize, as a lymphatic unit, a small group of lymphatic capillaries ending on the one hand blindly in the tissues, and on the other sending tributaries to collectors. These collectors may pass through small masses of lymphoid tissues on to the definite nodules known as lymph nodes or lymph glands. From these latter, trunks go to the venous system (Fig. 1). Fluids in tissue, originating from the blood, are returned thus to the blood stream.

Much of the difficulty in understanding the lymphatic system comes from the fact that it cannot be displayed by anatomical

methods ordinarily in use. Everyone who has studied the system by the necessary special methods is at once impressed with its tremendous extent. It is absent, it is true, from the eyeball, and lymphatics are not recognized in the brain. Further, lymphatics in the nerves are supposedly absent. With such exceptions, the lymphatics are almost universally found in the body. It is probably well to consider further some of these apparent discrepancies.

While it may be said that there are no lymphatics in the brain, and while there can only be loose analogies drawn between the anatomy of the subarachnoid space and that of the general lymphatic system, Mortensen and Sullivan[2] have shown by experiment a very interesting relationship.

These two workers, using the dog as their experimental animal, were able to demonstrate a ready communication between the cerebrospinal fluid system and the lymphatic system. Their work seems to be of sufficient importance to otolaryngologists to be briefly summarized here. Cisternal puncture was made in a dog and a small amount of the clear cerebrospinal fluid was permitted to escape. Two cubic centimeters of colloidal thorium dioxide was replaced. At the end of 30 minutes the convolutions of the brain were clearly outlined in x-ray films, the optic nerve was plainly seen, and the deep cervical lymph nodes could be differentiated from the neighboring tissues. Also the nasal area contained thorium, as shown by the films. In 90 minutes the cervical nodes were infiltrated as far as the third cervical vertebra and the submaxillary nodes were easily recognized. By the end of 6 hours it was easy to recognize the vessels entering the nodes and this picture remained practically stable for 2 months. Their studies showed that the deeper nodes were filling by way of the nasal cavity. This has been shown before, but not so clearly in the living animal. To quote from Mortensen and Sullivan, "The mass passes from the subarachnoid space along the olfactory nerves to the nasal mucosa and then by way of lymph vessels along the pharynx to the upper deep cervical nodes. The submaxillary nodes are filled by vessels running subcutaneously." Flow in the reverse direction was demonstrated by W. E. Le Gros Clark.[3] Clark demonstrated the movement of fluid along the perineural spaces of the olfactory nerves. After dropping potassium ferrocyanide and an iron ammonium salt into the nasal cavity of rabbits, he was able, one hour later, to identify Prussian blue in the meningeal spaces. It should not be concluded, however, that this means there is an actual anatomical communication with the spaces.

In this brief summary, other systems of pathways for communication between the nose and the central nervous system cannot be indicated; enough has been said to indicate that the lymph pathways and tissue spaces present at least one possible mechanism. The physiological continuity of the subarachnoid space with the nasal lymphatics has also been demonstrated by a number of other investigators. Much more remains to be done before the specific pathways of transmission for any disease can be considered solved.

In connection with the nervous system, a reference should be made to the apparent predilection of certain infections, such as rabies, for nerve trunks; and to the coursing of experimental injections along nerve trunks. While no lymphatic vessels are recognized in nerve trunks, it is possible either in living or preserved nerves to inject dyes into the nerve trunk and to follow the path for several inches. I have also followed procaine solutions along nerve trunks, although they are somewhat difficult to identify by color reactions in extreme dilutions. Among others, Sullivan and Mortensen[4] have demonstrated the flow of brominized oil along the sciatic nerve. Colored brominized oil in the dog will travel from the sciatic nerve to the subarachnoid space in 5 or 6 hours. This pathway is probably a tissue space and not a lymphatic, but this does not detract from the interest of the observation.

No doubt other peculiarities and interrelations between the lymphatic system and fluid spaces will be brought out later in the discussion of the labyrinth.

In turning to what is conventionally referred to as the lymphatic system, I shall briefly mention methods commonly used in its exploration and demonstration. Probably the two most frequently used, and most productive methods, are the injection of metallic mercury and the injection of an ethereal suspension of an oil paint. The latter is used in a mixture known as Gerota's fluid. In introducing the injection mass into the vessels, the method of random puncture, which consists of plunging a fine glass needle into the skin or serous or mucous surfaces, is used. If mercury is used it is allowed to flow in by gravity; Gerota's solution is introduced under the pressure of a small syringe. The random puncture method depends upon the fact that the lymphatic capillaries are opened by the needle and that the material finds its way from them into the larger trunks. In using glass cannulæ, I always prepare a number and try first one and then another until I find one that works well. The jagged, irregular character of the tip seems to be important. In the use of steel needles, I employ a trick that I

learned from Dr. William Snow Miller; he uses an old rusty needle with a still open lumen. This seems to tear the tissues quite as well as the glass needle. It is possible in the case of slightly larger lymph vessels to introduce a cannula directly. This calls for the greatest precision in the selection of pointed needles and a more precise technique.

The application of the random method of injection to the living, by Hudack and McMaster,[5] is interesting. By raising an intradermal wheal in dogs by injecting a dye, they followed the course of the subcutaneous lymphatics for a considerable distance in a few seconds. Personally I have repeatedly seen this injection dem-

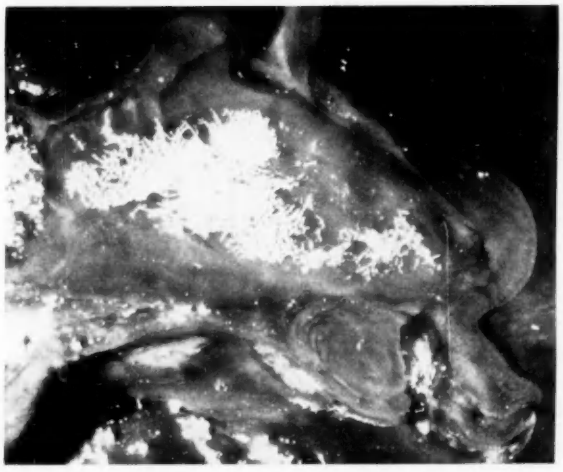

FIG. 2 — (Batson) — The lymphatic network of the mucous membrane of the nasal septum of an infant displayed by the injection of metallic mercury. This injection, obtained by the method of random puncture, illustrates the extensive nature of this system.

onstrate the lymphatics for a distance of 6 inches from the point of injection in the dog in less than 9 seconds. In my own forearm, and in the forearm of a student, I have followed the lymphatic course for 2 cm. in less than 5 seconds. In the human it is difficult to follow the vessels for a greater distance because of the thickness of the skin. As pointed out by Hudack and McMaster, the relation of this observation to the generalized reactions following diagnostic intradermal injections is most significant.

Among the anatomical methods, the method of random injection of metallic mercury has the advantage of beautifully displaying the capillary network, but it has the disadvantage of over-dis-

FIG. 3 — (Batson) — The lymph collectors of the head drain into a series of lymph glands having a ring-like distribution at the junction of the head with the neck. This may be called an occipitomandibular ring. From this series of nodes connections are made down the neck through a double chain of nodes.

tending the capillaries and making their differentiation from the collecting trunks very difficult. The early work of the French school was largely done by this method and many of the beautiful figures we see in our books today are copied from the plates of the early French investigators. After introduction of the ether color mass by Gerota, this method came to replace the mercury injection in the French laboratories. By this method the capillary plexuses are not so completely distended, and due to the ether, the fluid runs a long distance and is found coloring the collecting trunks and the lymph nodes. Specimens prepared by the two methods are necessary to display the total extent of the lymphatics of the head and neck. In the head and neck there are three features of the lymphatic system which should interest the otolaryngologist. First may be mentioned the extensive lymphatic network which is found in all the areas in which the otolaryngologist is concerned (Fig. 2).

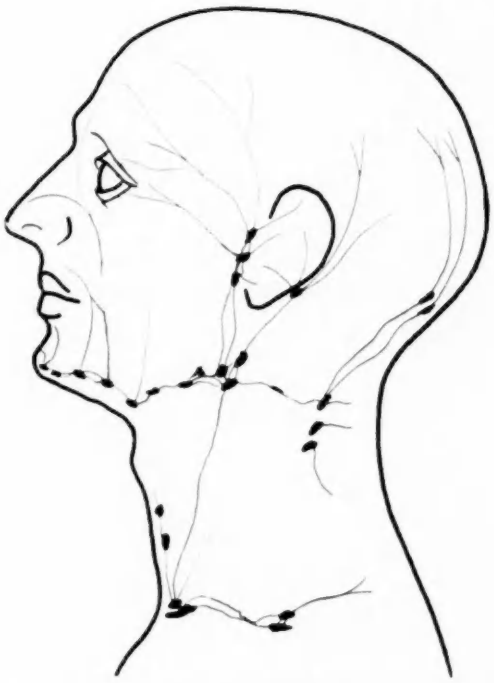

FIG. 4 — (Batson) — The lymphatic drainage of the various superficial regions of the head and neck to lymph glands is indicated.

These capillaries are immediately connected to the ring-like mass of lymph nodes, situated at the level of the occiput and the mandible. From here a chain of lymphoid tissues, both lymph nodes and lymph vessels, roughly divisible into superficial and deep portions, runs from the occipitomandibular ring to the veins at the root of the neck.

These general facts can best be summarized by reference to three figures. Figure 3 shows the distribution of the principal lymph nodes of the head and neck and, while the ring is not indicated as such and while the two columns of deep chains are not too obvious, the basis for the terminology is clear. The principal drainages in the head and neck area are indicated in the two summarizing figures (Figs. 4 and 5). One shows the drainages from the external areas and one the drainages from the mucous membranes and from the teeth. The form of these figures was inspired by two figures in Tandler.[6] The information contained, however, has been gathered from various sources, including personal observations.

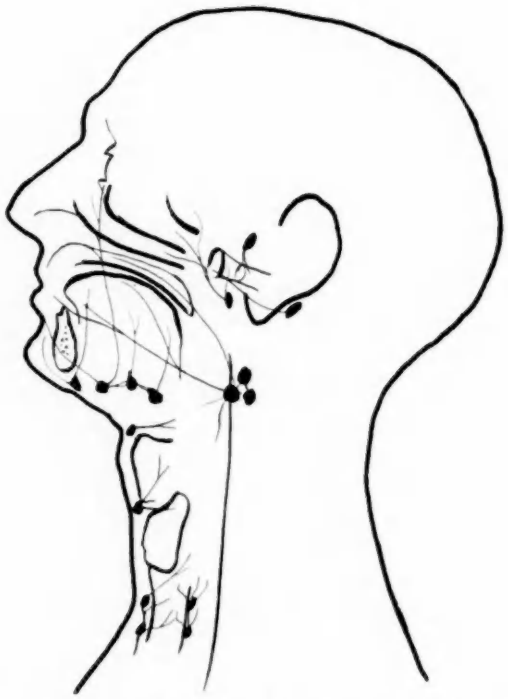

FIG. 5 — (Batson) — The drainage of lymph from the nose, throat, and ear are indicated in this diagram. The retro auricular connection of the eustachian tube is especially interesting.

Our last concern is the mucous membranes which come under the direct observation of the clinician, and which he penetrates in many operative procedures. In examining the appended figures (Figs. 6 and 7), one is almost led to wonder where there is room for the characteristic tissues of the parts. These specimens have been obtained by the use of mercury injections which result in an over-distension of the capillary network. However, the figures well indicate the universal character of the distribution of the lymphatic capillary bed.

In conclusion, it is obvious that we must further investigate the exact manner of the connections between the subarachnoid space and the lymphatic system. Further, more study is required of the drainage of some areas such as that of the eustachian tube. The physiologist must tell us more about the conditions that produce lymphedema. Finally, we should remember that every time an injection is made into the tissues, it is made potentially directly into the lymphatic stream.

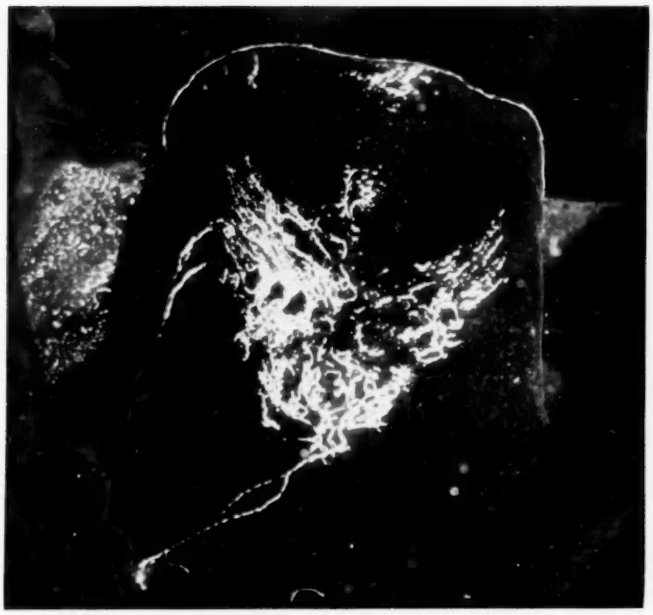

FIG. 6 — (Batson) — The lymphatic capillary network of the dorsum of the tongue. Mercury injection. Only the collectors to the left side were displayed. The incompleteness of this specimen shows why so many injections must be made in studying an area.

FIG. 7 — (Batson) — The richness of the lymphatic capillary distribution in the mucosa of the lateral nasal wall of an infant. This can be taken as representative of the distribution in the adult. Obviously, any surgical procedure within the nose must cut across many lymphatic capillaries.

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LYMPHATIC DRAINAGE OF THE HEAD

* * *

LYMPHATIC DRAINAGE OF THE LABYRINTH
WITH CLINICAL COMMENTS

A. G. POHLMAN, M.D.
BY INVITATION
OMAHA

THERE WAS A time when the lymphatic apparatus was regarded as directly continuous with the tissue spaces, and was held to be of great importance in the elimination of waste products resulting from tissue metabolism. The evidence, at the present time, indicates that lymphatics are blind-ending tubes lined with an endothelium, perhaps even a specific type of endothelium, which is structurally and functionally interposed between the tissue liquid and the lymph proper. The chances are that particulate matter is normally carried into the lymphatic by means of active agents such as macrophages. Under conditions of pressure, or where there has been tissue destruction, the wall of the lymphatic may be broken. The tissue juice, which affords the watery environment about the body cells, is quite completely under control of the capillary bed. If this were not true it would be difficult to account for local edemas which may arise without demonstrable mechanical or infective cause.

While the tissue spaces are ordinarily more or less microscopic clefts, in certain situations they may be well developed and contain an amount of liquid which is entirely out of proportion to the numbers and the activity of the cells to which it is applied. It is not quite fair to call such liquid "lymph" any more than one may regard the cerebro-spinal liquid as "lymph."

The subarachnoid space, for example, arises through a delamination of the connective tissue surrounding the central nervous system, and so far as I am aware, its development is not comparable to that of true lymphatics. Neither does the finished product conform with the structural characteristics of the lymphatic system. While some of the contained liquid is absorbed directly into

the capillary bed of the pial vessels, a great part escapes from the subarachnoid space into the venous return through the arachnoid granulations. There is no definite evidence that much of it is returned through lymphatics.

When the trunks of the cranio-spinal nerves leave the territory of the dura, the meninges are carried out with them to become continuous with the perineurium. In the case of the optic nerve, the subarachnoidal cleft may be demonstrated as far as the eyeball, which is, of course, what might be expected because the optic nerve is in reality a brain tract. Other extensions of the subarachnoidal cleft have been described along the course of true cranial nerves, and in particular, the one associated with the glossopharyngeal nerve is important to our discussion.

The internal ear presents another of these relatively large tissue spaces where the membranous labyrinth is dissected away from the surrounding otic capsule through the development of the perilymphatic spaces. Streeter has studied this problem in great detail and arrived at the conclusion that the development of the perilymphatic space is analogous to that of the subarachnoid space, as described by Weed. He also found evidences of a developmental prolongation of the subarachnoidal cleft along the course of the glossopharyngeal nerve, and makes the following notation: "The communication that is found in the adult between the Scala tympani and the subarachnoidal space in the neighborhood of the Fenestra cochleæ, the so-called Aquæductus cochleæ, is established quite late. In fetuses of 85 mm. crown-rump length, it exists as a tubular pouch projecting from the arachnoidal space along the glossopharyngeal nerve toward the Scala tympani. In the 130 mm. fetus, the oldest examined, the pouch is longer and nearly reaches the Scala."

The evidence is that a tissue cleft is prolonged from the subarachnoidal space along the glossopharyngeal nerve to follow the venous return from the cochlea toward the Scala tympani, and the inference is that it may form an open communication between the subarachnoid space and the perilymphatic apparatus (a true Ductus perilymphaticus). This is interesting morphologically because this type of development is not characteristic for lymphatic channels, and, further, whether the communication is a tissue cleft or a true lymphatic, its presence might constitute a pathway through which infections could pass in either direction. Indeed such a pathway might be even more dangerous if it were a tissue cleft rather than a lymphatic channel where the continuity would be broken by the limiting membrane of endothelium.

Retjö, who has done much work on the morphology of the labyrinth, particularly in reference to his conception of auditory mechanics (to which I do not agree), feels that this channel can not be an open communication. He cites, in confirmation of his views, the fifty-five cases of suppurative labyrinthitis reported by Knick, where the cerebro-spinal liquid was normal.

Karbowski finds as a result of injection experiments that the Aquæductus cochleæ is an open communication between the perilymphatic space and the subarachnoid cleft in guinea pig, dog and rabbit and states it is variable in the human being. This author also finds evidence that injected material may follow the course of the Nervus tympanicus toward the subarachnoidal expansion about the glossopharyngeal and believes it may be a route followed by infections of the middle ear toward the meninges. I have no information on the frequency with which this may occur. Chilow, employing a modified Bezold labyrinth manometer, did not find evidences of an open communication.

Refinements in injection technique have made certain observations possible but these results must be accepted with reservations. For example, Yuien reports that according to his results the flow of the lymphatic in a nerve trunk is in a direction contrary to that in which the stimulus is carried. This is an interesting observation which I would like to see confirmed before I would care to discuss it. By the same token, we are by no means certain that injection experiments may not produce pressure dissections which result in the formation of artificial tissue spaces comparable to some of the so-called fascial compartments. If I remember correctly, one investigator of the sympathetic system found the more decomposed the tissue, the more readily the injection followed the distribution of the nerve studied. It must also be borne in mind that injection experiments with particulate matter do not necessarily indicate the course taken by infections even when accompanied by tissue destruction.

Quix and von Egmond realized the many valid objections to the injection technique as usually performed. Personally, I regard Quix as one of the outstanding investigators, even if we do not see eye-to-eye in matters pertaining to audiometry. These observers devised an ingenious method of instilling a mixture of an iron salt and cocaine into the middle ear region. They timed the diffusion of the iron in terms of the anesthetic effect of the cocaine, and precipitated the iron salt in situ by means of the familiar Prussian blue reaction. They found in the living animal (guinea pig) that

the iron salts did not pass into the internal ear through the round window membrane but diffused directly through the bony wall toward the base of the skull, toward the cranial cavity, and directly through the wall into the semicircular canals and cochlea. It was only after the salts had passed into the cranial cavity that traces were found in the Aquæductus cochleæ and then only in a restricted portion of the cranial end of that pathway. The color reaction did not occur in the Stria vascularis or in the cochlear ganglion and nerve. It appeared that the iron salt passed through the bony partitions between the cochlear turns toward the base of the skull. Neither Ductus nor Saccus endolymphaticus showed traces of the precipitate.

In dead animals, however, a similar technique showed different results. Here the color, instead of passing through the bone, entered the cochlea through the round window membrane. The dead animals showed traces of color reaction in the Stria vascularis and also in the cochlear ganglion and nerve.

The failure of the iron salt-cocaine mixture to pass through the round window in the living guinea pig is not in accord with the experimental evidence on the application of cocaine to the round window membrane in animals studied with the Wever-Bray technique. In any event the work of Quix and von Egmond points out that living animals must be studied, and that the information obtained from dead animals must be accepted with reservation.

I had hoped to be in a position where I might contribute some experimental evidence of my own, but obsession in problems of audition made this impossible. However, this is what I had in mind. It is known that when particulate matter is injected into the tissue spaces of a living animal, it is promptly engulfed by macrophages. These macrophages migrate toward and enter the lymphatic channels. It should, therefore, be possible to culture marked macrophages elsewhere in the body and then introduce these cells into the perilymphatic spaces. These marked cells should then tend to migrate and make it possible to identify the drainage systems under relatively normal conditions. Whether this may be possible remains to be seen.

It must be confessed that the entire problem of relation of perilymphatic spaces to subarachnoidal space is more or less obscure. The clinical indications, however, are quite clear, and I see no reason why more definite information on the anatomical relations will necessarily influence a proper surgical treatment which has been based on shrewd observation and long clinical experience.

LYMPHATIC DRAINAGE OF THE HEAD

III

LYMPHATIC PATHWAYS FROM THE NOSE*

OLOF LARSELL, Ph.D.
BY INVITATION

PORTLAND, ORE.

IN A SERIES of papers beginning in 1919, Mullin[1,2,3,4] has called repeated attention to the relation between drainage from the paranasal sinuses and certain types of chest infection. He pointed out that particles of India ink or bacteria introduced into the sinuses of cats and rabbits, the sinus membranes being injured to facilitate absorption, were taken up by the lymphatics and found their way to bronchial and mediastinal lymph nodes. Mullin and Ryder[5] reported that inhalation of carbon suspensions or of tubercle bacilli by rabbits results in pigmentations or tubercular lesions similar to those of human tuberculosis, including involvement of bronchial lymph nodes.

Various clinical observers have emphasized one or the other possible routes of entry of infectious material to lungs and related lymph nodes in relation to bronchitis, bronchiectasis and other pulmonary disturbances. Quinn and Meyer,[6] Clerf,[7] and others have pointed out the significance of aspirated material from the sinuses. Lawson,[8] Mullin and others have written on the significance of the blood stream or combined lymphatic and blood stream routes.

Too little attention has been paid in all these studies to the defense mechanism of the lung itself and to the arrangement of lymphatics and lymphoid tissue therein in relation to the possible pathways of entry of particulate or infectious material. The beautiful and detailed studies of W. S. Miller[9,10] on the lymphatics of the lung give a firm basis on which to build a more complete knowledge of the pathways of infection in the lung and neighboring structures.

* Research authorized and financed by the Council of the American Academy of Ophthalmology and Otolaryngology, from the Departments of Anatomy and Otolaryngology, University of Oregon Medical School.

During recent years much attention has been given to the lining of the pulmonary alveoli. Histologists are divided between the conception of an entodermal epithelial lining of the air-sacs and the views advanced by Fried[11] and others that the lining is made up of mesodermal phagocytic cells, closely related if not identical to the histiocytes and macrophages elsewhere in the body. According to some views this lining is not continuous. Rose[12] holds that the pulmonary capillaries themselves form the immediate lining of the air spaces, the latter thus corresponding to an open wound. Loosli[13] has presented evidence that the capillaries are uncovered. Miller[14] and others, on the other hand, have provided new evidences of a layer of flattened cells between capillaries and alveolar spaces. Fried has strongly emphasized the protective function of the alveolar surface against bacterial invasion.

Without attempting to determine the definitive structure of the alveolar wall, experimental evidence will be presented in the following description regarding the portals of entry of particulate matter into the lungs and the pathways of drainage from the paranasal sinuses and related regions into the chest. It will be attempted to correlate the evidence presented with the recognized facts of pulmonary anatomy.

MATERIAL AND METHODS

Eighteen adult cats and three kittens were used in the experiments to be described. They were anæsthetized with Nembutal, given intraperitoneally, or with Dial (Ciba) given intramuscularly. These anæsthetics were used, in preference to ether, in order to prevent the large amount of mucous secretion in the air passages which results from the use of ether in cats, and also because of the greater ease of administration. The adult animals were kept under the anæsthetic for periods varying from thirty minutes to fifty-five hours before they were killed. During these varying periods the lymphatics related to the upper respiratory tract were subjected to continuous flow of the injection masses or to repeated injections with them at intervals of two to twelve hours.

The injection masses employed were of three different types, namely, Prussian blue, trypan blue and potassium ferrocyanide. Prussian blue is usually employed for the injection of lymphatics. Ordinarily it is used as a suspension in a thin solution of warm gelatine. We used, however, a suspension of artist's Prussian blue, obtained in tin tubes mixed with oil. This was dissolved in turpen-

tine in the ratio of equal parts of the mixture, as it comes from the tube, and turpentine. This suspension was then diluted with four to five volumes of ether and injected with a number 25 hypodermic needle and Luer syringe. The needle was pushed through the soft palate and into the posterior wall of the pharynx at various points. Some of the mass escaped into the pharynx, whence it would be removed with a swab. Most of it reached the lymphatic spaces and vessels, lymph nodes and fascial spaces of the retropharyngeal and cervical regions, as described below. Supplementary injections of the cervical lymph nodes, to fill their efferent lymphatics, were made by puncture of the nodes with the needle. After ten to fifteen minutes of injections with this mass the animals died, probably from the effect of the ether superadded to the Nembutal already administered. They were then dissected and placed in 10 per cent formalin solution. The Prussian blue was used to ascertain the gross distribution of lymphatic vessels.

A method which was designed to demonstrate finer lymphatics and lymph spaces and, it was hoped, would make unnecessary puncture of lymph nodes to demonstrate their efferent lymphatics, was used as follows: three per cent solution of potassium ferrocyanide was injected into the frontal sinus or into the posterior wall of the pharynx, under slight pressure, for periods up to fifty-five hours. This substance is relatively non-toxic and produced no ill-effect, so far as could be determined, on the animals anæsthetized with Nembutal or Dial. After varying periods the animals were killed, dissected as much as necessary, and the head, neck and thorax, with organs exposed, were placed into 5 per cent ferric citrate in 10 per cent formalin. The ferric citrate precipitated Prussian blue from the potassium ferrocyanide, in the lymphatics, lymph nodes and other structures which the injected fluid had reached. The formalin served to fix and preserve the tissues. To prevent the entrance of potassium ferrocyanide into the air passages and lungs during injection a tracheal cannula was inserted at the beginning of most of these experiments. When the animal was killed the lungs were filled with fixing fluid through the cannula.

A third procedure consisted in introducing trypan blue solution in normal saline under the mucosa of the frontal sinuses or posterior pharyngeal wall. It will be recalled that trypan blue forms a colloidal solution, the particles of which are ultramicroscopic. Only when stored in phagocytic cells do the particles accumulate sufficiently to become visible with the microscope. Two methods

were used with the frontal sinuses. One consisted in exposing the bony wall above the eye, chipping away the bone to expose the membrane and then fixing needles in position between the membrane and bone with bone wax. The needles, attached to flexible rubber tubing, were then connected with a funnel containing trypan blue. The solution was allowed to drain into the sinus membrane at the pressure produced by raising the funnel 50 mm. above the animal. A second method consisted in inserting small glass cannulæ through small corks fitted into trephine openings in the bony roof of the frontal sinus and allowing the solution to enter the sinus itself under pressure similar to the above during a period of many hours. The ostium of the sinus had previously been plugged with cotton wool and bone wax.

To inject the posterior pharyngeal wall needles of suitable length and size were thrust through the soft palate and into the membrane of the pharynx. Adult cats under anæsthesia and fitted with tracheal cannulæ were injected at intervals of about two hours for twenty-four to fifty-five hours. Two kittens, anæsthetized for the first injection, were injected two to three times per day for eight and one-half days and fourteen days, respectively. These did not have tracheal cannulæ, so some of the trypan blue reached the trachea and probably the lungs by overflow dripping from the pharynx. A third kitten was subjected to repeated injections of trypan blue directly into the trachea for four days.

Sections were made of cervical lymph nodes, carotid sheath, œsophagus, portions of the neck, lung, bronchial and mediastinal lymph nodes, etc. They were stained with Orange G or with safranin. The latter has the advantage of staining both nuclei and cytoplasm.

DESCRIPTIVE

The lymph drainage from the nasopharynx in the cat is chiefly toward the retropharyngeal and laryngeal region. Three cats given repeated injections of potassium ferrocyanide under pressure into the nostrils over a period of two hours showed an abundant precipitate of Prussian blue in the cortex, and some in the medulla, of the deep cervical lymph nodes. The small lymph nodes, follicles and spaces of the retropharyngeal region also showed precipitate, and a small amount was present in the region of the palatine tonsil, especially in the spaces and channels of the muscle behind the tonsil. These cats were each fitted with a tracheal cannula and the trachea was tied off anterior to the cannula to prevent the fluid

from diffusing out in the visceral cervical space. In two of these cats the tracheal lymph ducts showed precipitate.

Cat 9, injected with potassium ferrocyanide into the frontal sinus for eight hours by the procedure above described, showed blue precipitate in the deep cervical lymph nodes, in the retropharyngeal region, and in bronchial and mediastinal lymph nodes, on microscopic examination. A tracheal cannula had not been thought necessary in this animal. The mucosa of the trachea and bronchi showed considerable precipitate, after fixation, indicating escape of potassium ferrocyanide into the air passages during the period of injection. The precipitate in the bronchial, and possibly in the mediastinal lymph nodes may therefore have been due to absorption of the injected solution by the lymphatics of the lungs.

It is clear from these experiments that the lymphatic drainage from the nasopharynx in the cat is primarily into the retropharyngeal and deep cervical lymph nodes. The drainage from the frontal sinuses is also into the retropharyngeal and deep cervical lymph nodes. The retropharyngeal nodes in the cat are small, most of the lymphoid tissue of this region being in the form of follicles rather than nodes. The normal membranes of the frontal sinuses are so thin as to leave little room for lymphatic vessels. There are, however, lymph spaces which gradually converge toward the main blood vessels supplying the sinuses. Around these vessels there are small lymphatic plexuses which drain into the retropharyngeal region and, directly or indirectly, into the cervical or laryngeal lymph nodes.

The efferent drainage from the laryngeal lymph nodes is through the tracheal trunks on the respective sides of the trachea. They drain into the corresponding jugular vein at its junction with the subclavian vein. These ducts receive tributaries from the posterior wall of the visceral space of the neck, as is shown by puncture injections of this wall at various points. These features are readily demonstrated by injections with the turpentine solution of Prussian blue, if supplemental injections of the cervical lymph nodes are made to fill the larger ducts.

Injections of Prussian blue into the posterior wall of the pharynx, using some pressure, result in an apparently continuous mass of blue color from the laryngeal region, posteriorly, on each side of the trachea, to the base of the neck. This continuous mass is formed by confluence of the tissue spaces in the loose areolar tissue along the œsophagus and trachea. The injection mass reaches these spaces from the tissue spaces of the pharyngeal wall, when

FIG. 1 — (Larsell) — Portion of dorsal wall of mediastinal œsophagus of cat (number 16) injected with trypan blue repeatedly for fifty-five hours into mucous membrane of posterior nasopharynx. Some of the trypan blue laden macrophages are indicated by arrows.

introduced into the retropharyngeal region, and thus extends caudally to the anterior boundary of the chest. A similar but smaller mass of injected material extends downward in the carotid sheath.

In the fibrous coat of the posterior wall of the œsophagus there are tissue spaces which extend, by continuity one with another, to the mediastinum. Injections with trypan blue show numerous macrophages along the length of the dorsal part of this tube as far as the upper mediastinum (Fig. 1). The lateral and ventral œsophageal walls do not show such cells. Puncture injections of Prussian blue into the dorsal wall of the visceral cervical space show some lymphatics which drain into the tracheal ducts, as stated above, but a gentle, steady pressure on the syringe gradually fills tissue spaces which extend downward toward the base of the neck.

By puncture injection into the dorsal wall of the space in the base of the neck, about one inch above the anterior wall of the mediastinum, the mediastinal tissue spaces, dorsal to the heart and lungs, were filled on each side. The injection mass extends well toward the bronchi and the hilum of the lung, on the one hand, and well toward the caudad part of the mediastinum on the other hand.

Cats 11 and 12, fitted with tracheal cannulæ and injected with trypan blue for twenty-eight and twenty-two hours, respectively, by inserting a needle into the posterior wall of the pharynx, showed trypan blue in sections of the laryngeal nodes, small deep inferior cervical nodes, bronchial and dorsal mediastinal nodes. In the latter there are numerous granules both in peripheral and deep sinuses and in large phagocytic cells in the nodes. The reticular cells of cervical, bronchial and mediastinal nodes also show ingested trypan blue. The septal and dust cells of the lungs and the pulmonary lymphatics and lymph follicles also show the dye.

Cats 13 and 14, both kittens about six weeks old at the time of initial injection, received doses of about 1 cc. of 0.5 per cent trypan blue solution injected several times per day into the posterior pharyngeal wall for fourteen days and eight and one-half days, respectively. They suffered no untoward effects save for five to ten minutes after each injection, during which time they were removing the overflow of the injection from the nasopharynx. Some of the excess undoubtedly dripped into the trachea and the lungs.

These kittens, when killed and dissected, showed substantially the same picture. The deep cervical or laryngeal nodes were strongly colored with trypan blue, visible grossly. The prevertebral fascia showed blue coloration, grossly, about midway to the base of the neck. The amount of coloration diminished downward, with but a small amount in the lower part of the neck region.

Microscopically the upper part of the œsophagus showed great numbers of blue laden macrophages in the loose connective tissue of the dorsal wall and extending into the striated muscle layer along the connective tissue septa. Between the muscle bands, in the thinner septa, blue laden cells are numerous throughout the muscular layer. The connective tissue of the dorsal œsophageal wall also shows small lymph channels, in sections, containing lymphocytes and blue laden phagocytes. Evidently there was much diffusion of the injected solution downward from the posterior pharyngeal wall along the tissue spaces behind the œsophagus. Migration of histiocytes and macrophages probably does not account for the widespread distribution of the trypan blue along the

dorsal œsophageal wall and caudally toward the thorax, since the lateral œsophageal walls are almost free of blue granules in corresponding cells.

In a mediastinal lymph node dorsal to the œsophagus, which was dissected out, together with neighboring adipose tissue and fascia, there are numerous blue laden macrophages. Between the fascial layer and fat there is a lymphatic vessel of considerable size containing numerous lymphocytes and dye laden macrophages. This is probably an afferent lymphatic, as the mediastinal node described is regarded as having received its trypan blue from the mediastinal and lower cervical lymph spaces, rather than from the lungs, by reason of its position and apparent connections.

Cat 16, fitted with tracheal cannula and kept under anæsthesia for fifty-five hours, was given repeated injections, during this period, of trypan blue, under pressure, into the posterior pharyngeal wall. The needle was kept attached in position between injections and was continuously filled with the solution.

Grossly the cervical lymph nodes are colored a deep blue by the dye. From the posterior pharynx a zone of deeply stained tissue extends along the prevertebral fascia into the mediastinum and thorax, diffusing into the loose connective tissue on each side of the œsophagus. Behind the œsophagus there are definite lymphatic channels as far as the inferior cervical region. Within the upper thorax the blue coloration extends beneath the parietal pleura for a short distance on each side. The dorsal mediastinal lymph nodes also appeared blue to the naked eye. The upper mediastinal œsophagus shows lymph channels with blue coloration in the adventitious coat just outside the muscle layer.

Microscopically both the lower cervical and mediastinal œsophagus shows trypan blue laden macrophages in the adventitious coat and also in the septa extending into the muscle layer. Lymph channels containing lymphocytes and trypan blue laden phagocytes are abundant. These discharge from the mediastinal œsophagus into mediastinal lymph nodes. The lower thoracic œsophagus and aorta show no indication of the trypan blue. The superior mediastinal nodes show peripheral sinuses distended with fluid and containing many trypan blue laden macrophages. Such cells are less numerous in the deep part of the nodes. The carotid sheath, both upper and lower, has spaces filled with trypan blue laden phagocytes. There are also definite lymphatic channels distended with fluid and containing phagocytes and lymphocytes. The prevertebral

fascia of the base of the neck microscopically shows lymphatic vessels and loose connective tissue containing numerous phagocytes filled with trypan blue.

THE LUNGS AND BRONCHIAL LYMPH NODES

In a number of the experimental cats the bronchial lymph nodes show precipitate from the potassium ferrocyanide and ferric citrate treatment or trypan blue granules. It is necessary to determine the pathway of entrance. There is some intercommunication between tracheo-bronchial and mediastinal nodes but the bronchial nodes near the hilum of the lung are usually regarded as receiving their afferent lymphatics from the lungs. The detailed anatomical studies of W. S. Miller[9, 10] have provided very complete information concerning the lymphatic drainage of these organs.

Cats 13 and 14 were injected with trypan blue over relatively long periods without tracheal cannula. There was, therefore, some dripping, very probably, as already stated, from the pharynx into the lungs. Yet the trachea and bronchi were quite clean when the animals were killed several hours after the last injection. The lungs of these kittens were blue macroscopically and on examination of sections they were seen to be filled with cells of various kinds, laden with trypan blue granules. The air-sacs contain numerous large cells, some free, other in various stages of detachment from the alveolar walls. Very many of them contain trypan blue. The peribronchial and perivascular lymphatics in all parts of these lungs are distended with fluid and contain numerous phagocytic cells with trypan blue granules (Fig. 2). The lymphatics of the pleura likewise are distended and contain phagocytes. It will be recalled that the lymphatic drainage from the outer part of the lung is into the pleura.

We shall not enter here into a discussion of the lining of the air-sacs. Histologists are divided into two schools, some holding to the view that the alveoli of the lungs are lined with flattened entodermal cells, while others claim that the lining, so far as it is present, is mesodermal and related to macrophage cells. A recent brief discussion of the salient points of the opposing views is contained in the new (1936) edition of Bremer's *Text Book of Histology*,[15] to which reference is made.

The point of importance in the present presentation concerns the pathway of entrance into the lung of the trypan blue granules

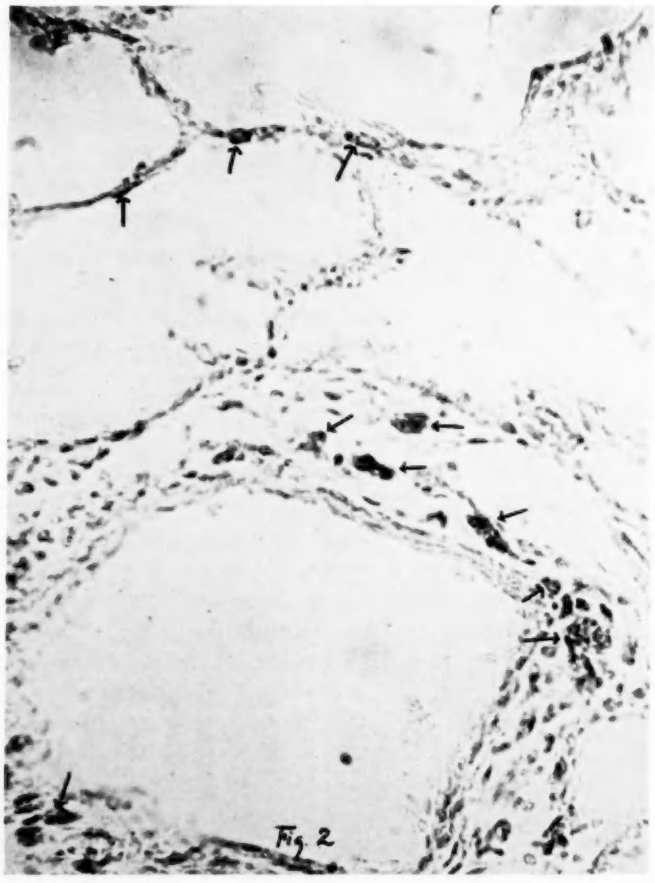

FIG. 2 — (Larsell) — Portion of lung of kitten (number 14) showing trypan blue laden cells in perivascular lymphatics and septal cells containing dye particles. This cat was injected with the trypan blue into the posterior pharyngeal wall at intervals for 8½ days. Some of solution escaped into trachea, probably at each injection.

contained in the septal and dust cells and in the phagocytes within the lymphatics. Since the only lymphatics connected both with the lungs and with adjacent structures are the efferents at the hilum, there remain but two possible pathways of entry of the phagocytosed material, namely, the trachea and the blood stream.

In Cats 13 and 14 both these channels were open. Both of these animals show a very large amount of phagocytosed material within the lungs. This may be due to the length of the experiment with

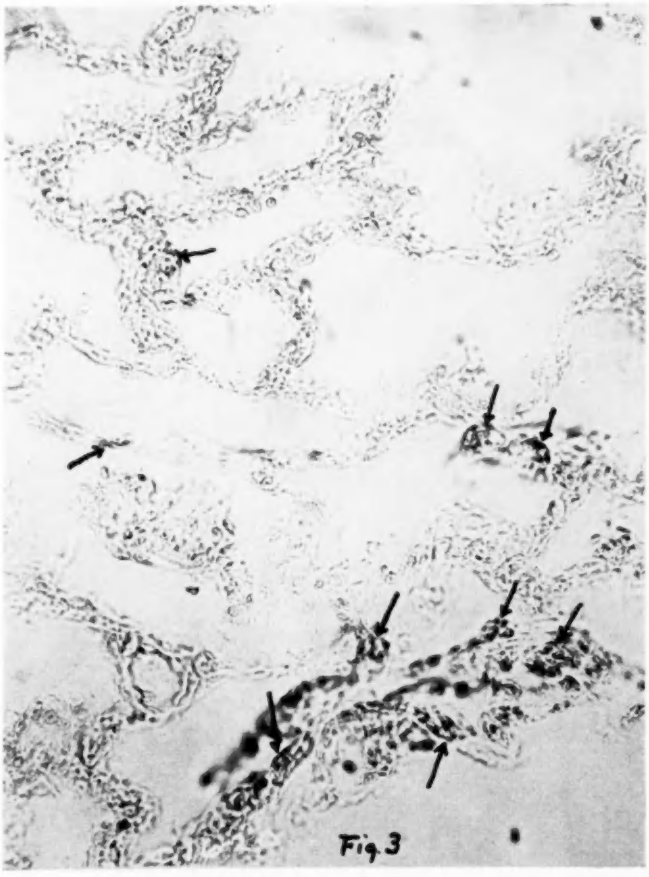

FIG. 3 — (Larsell) — Portion of lung of cat (number 16) provided with tracheal cannula and injected repeatedly for 55 hours with trypan blue into posterior pharyngeal wall.

them, to the fact that both channels of entry were open, or to a combination of these two factors. It was easy to rule out the tracheal pathway by repeating the experiment on animals provided with a tracheal cannula. A number of cats were so treated. It was thought desirable to keep them anæsthetized during the experiment, so it was not practicable to continue the experiment longer than twenty-eight to fifty-five hours. The results were essentially the same in all, save for the quantity of trypan blue encountered within the lungs. Since Cat 16 was continued for the longest period, namely, fifty-five hours, the more detailed description will be

based upon the results obtained with it. In this animal the lungs showed peribronchial and perivascular lymphatics filled with trypan blue laden phagocytes (Fig. 3). The pleural lymphatics also contained such cells. Neither pleural nor intrapulmonary lymphatics were as distended with fluid as in the kittens above described.

Septal cells and dust cells containing trypan blue are very numerous, but the amount of blue in the individual cells and the size of the granules is less than in the kittens. This is the only difference clearly marked, although one gains the impression that the number of cells containing granules is not so great in the adult cats as in the kittens. This may be due to differences in the time involved, to the closure of the tracheal portal of entry in the adult cats, or to both factors. There is, however, no question of the presence of granules in the phagocytic cells of the cannulated cats.

The trypan blue could have entered the lungs only through the blood stream in these animals, taking its path from the retropharyngeal and cervical lymph nodes, through the tracheal lymph ducts to the external jugular veins, and thence to the right heart and the pulmonary capillary bed. Diffusing through the endothelial cells, the colloidal particles were taken up by the septal cells. By repeated performance of this phagocytic function on the part of these cells, the ultramicroscopic colloidal particles gradually accumulated within the cells sufficiently to become visible granules. The size of the granules within the cells in animals treated for different periods of time is roughly in proportion to the period of phagocytic activity in ingesting the trypan blue. They are largest in the animals subjected to injections for the longest period. Particles of trypan blue are found along the capillary bed in general, but also in detached cells in the alveoli. The endothelial cells are in such close juxtaposition to the flattened cells, be they epithelial or macrophages which cover the capillaries, that the technique employed is inadequate to determine whether such particles are contained within them or not. The preponderance of evidence is negative, in our preparations. Foot,[16] who has made a special study of phagocytic activity by the endothelium of the pulmonary bed has recently revised his earlier position and holds that it does not have phagocytic properties, a view shared by Wislocki.[17]

It is generally accepted that the so-called "dust cells" phagocytose particulate material brought to the alveoli with the air in breathing. Fried holds that the lining cells of the alveoli are macrophages, and that the alveolar wall "is a veritable fortress when

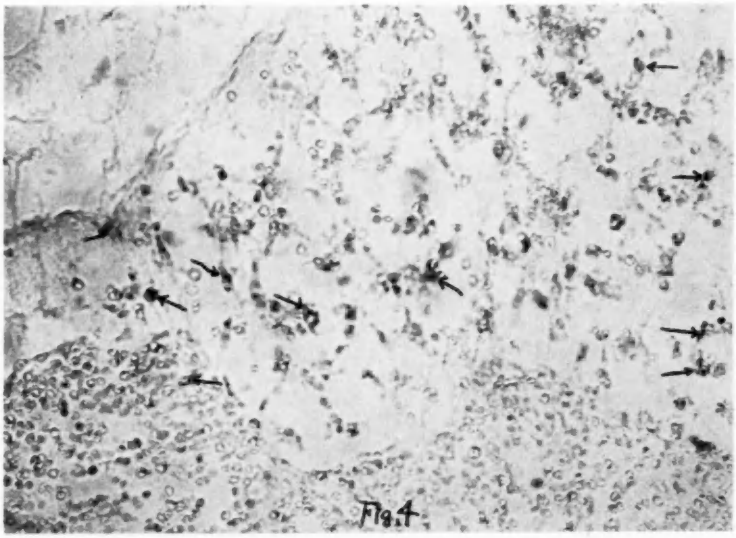

FIG. 4 — (Larsell) — Part of anterior mediastinal lymph node of Cat 14, showing trypan blue laden macrophages.

bacteria have penetrated into the respiratory portion of the lungs." He found that infection by the pathway of the pulmonary blood stream was much more easily produced than by the tracheal route. Wislocki has shown that carbon particles 1 to 3 microns in diameter injected into the blood stream of rabbits and cats were first deposited in the lungs as capillary plugs. They are then gradually removed through escape into the general circulation, through lymphatics draining into regional lymph nodes, and through phagocytosis by "clasmatocytes." The origin of the latter Wislocki was unable to ascertain.

We have seen a few capillary plugs of trypan blue in the lungs of our cats but they appear relatively unimportant. It seems more likely that the fine colloidal particles of this substance diffused through the endothelial cells, as they reached the lungs, from the beginning of the series of injections to which our animals were subjected. In this respect injections with trypan blue would differ from bacterial invasion of the lungs by the same route. Bacteria would probably form plugs, if present in sufficient number, as did the carbon particles of approximately comparable size (Streptococcus, 0.6 to 1.5 microns). The method of removal by phagocytic cells is the same for inert carbon particles and trypan blue, and

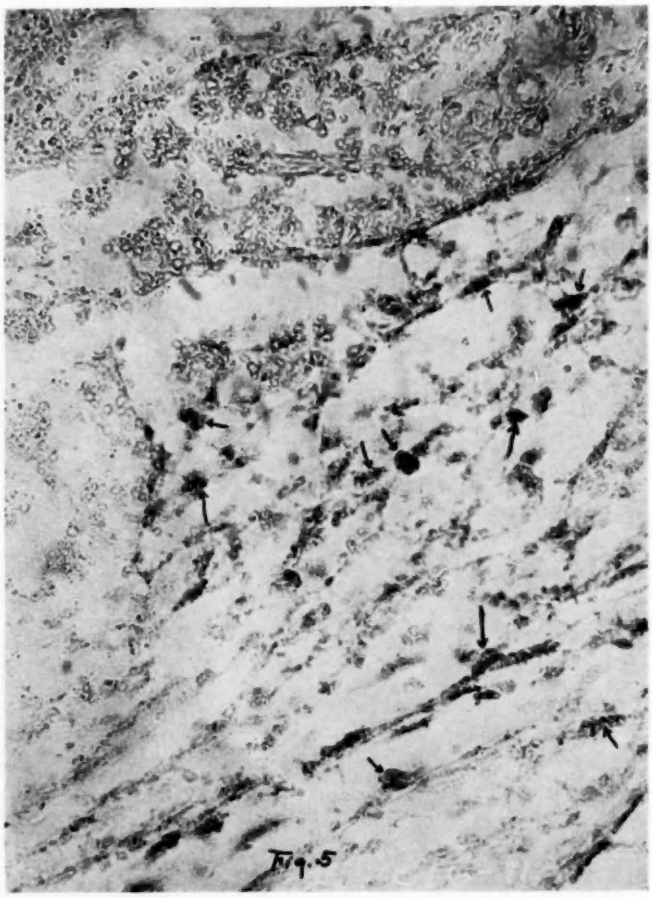

FIG. 5 — (Larsell) — Part of anterior mediastinal lymph node of Cat 16, showing trypan blue laden macrophages.

undoubtedly for bacteria, save that the latter through their proliferation and formation of toxic substances present a more serious problem to the defense mechanism.

As stated above, the trypan blue laden phagocytes leave the lung through the pulmonary lymphatics. Some of these drain directly to the hilum of the lung, accompanying the bronchial or blood vessel walls. From the outer portion of the lungs the drainage is first to the pulmonary pleura and then to the hilum through the pleural lymphatics. By either pathway the pulmonary lymph

FIG. 6 — (Larsell) — Part of tracheobronchial lymph node of Cat 16, showing trypan blue laden macrophages.

stream passes eventually into the tracheobronchial lymph nodes and neighboring nodes in the mediastinum. In all our animals in which the lungs showed trypan blue these lymph nodes were colored with the dye (Figs. 4, 5 and 6).

Some of the phagocytic cells also escape through the air passages. "Dust cells" laden with trypan blue are encountered free in alveoli in all parts of the lung, even those just beneath the pulmonary pleura. Such cells are found, mixed with mucus in the bronchi, in considerable numbers in some of the cats which had been cannulated. In many of the smaller bronchi of Cat 11, in spite

of tracheal cannula, the mixed mass of cells and mucus almost closes the lumen. In the larger bronchi they lie against the wall, enclosed by mucus. The presence of dye in their cytoplasm indicates that they must have become detached from the septal walls before the animals were killed. They obviously are desquamated cells which would normally have been discharged with the mucous secretions through the trachea. Cat 21 was injected on five successive days with about 1 cc. of trypan blue solution, introduced with a needle directly into the trachea below the larynx. The dye-containing dust cells are about as numerous as in cats whose bronchial passages were guarded by tracheal cannulæ. The septal cells with blue granules do not appear to be so widespread. Both peribronchial and perivascular lymphatics, however, are filled with trypan blue laden phagocytes. The larger bronchi show a considerable coating of trypan blue over the epithelial surface. The bronchial and tracheobronchial lymph nodes are blue macroscopically.

SUMMARY AND CONCLUSIONS

There are four routes by which material in solution or suspension can reach the bronchial and mediastinal lymph nodes from the paranasal sinus region. These are (1) the trachea; (2) the combined lymph node, tracheal lymph duct and blood vascular path, through the right heart and pulmonary bed; (3) the purely hematogenous path; and (4) lymph spaces and channels in the visceral cervical space, the dorsal wall of the œsophagus, the prevertebral fascia and related structures which communicate with the anterior part of the mediastinum.

The tracheal route is obvious. It is particularly important in air-borne infection. The experiments with trypan blue clearly indicate that drippings from the posterior pharyngeal wall and the nasopharynx also reach the lungs. The particulate matter, phagocytosed by septal and dust cells, is in part eliminated through the bronchial passages, but most of the phagocytic cells get into the perivascular, peribronchial and pleural lymphatics and thence into the bronchial and neighboring lymph nodes. Lymph follicles in the bronchial walls within the lung become enlarged, and when bacterial invasion is involved, no doubt become infected.

Colloid material reaching the lungs by the combined lymphatic and blood vascular route diffuses through the capillary walls to be phagocytosed by the same types of cells as serve for the tracheal route, namely, septal cells and the so-called dust cells. The pathway

of these cells from the lungs is again through the various lymphatic channels of the lungs to bronchial and mediastinal lymph nodes. When particulate matter of larger than colloidal size enters the pulmonary bed, there is formation of capillary plugs with subsequent phagocytosis. The phagocytes here involved also can escape from the lungs only by the lymphatic channels already named. Bacterial invasion undoubtedly may form capillary plugs which in turn become centers of proliferation and of long continued phagocytic activity, with long continued irritation to lymphoid tissue and neighboring structures.

The purely hematogenous route of invasion from the sinus region is possible, but appears quite unlikely. Aside from the mediation of lymph nodes and lymphatic vessels, the pathways would be the same as when the lymphatic route is involved. The pulmonary lymphatic pathways and cellular elements would, of course, also be the same.

The fourth route mentioned, namely, the lymph spaces and channels from the neck which communicate with the mediastinum, is probably of little significance. Bacteria escaping from the retropharyngeal region into adjacent tissue spaces are probably phagocytosed by the numerous histiocytes in the looser tissues before they have gotten far. The communication of tissue spaces in the connective tissues continuous from the neck to the thoracic wall and mediastinum, however, indicates the possibility of this pathway.

The lymphatic-blood vascular and the tracheal routes are certainly the most important paths by which material from the sinuses entered the lungs and related lymph nodes in the experiments reported. The evidence does not permit definite conclusions as to which of these two routes was the more important but appears to point toward the combined lymphatic and blood stream path.

* * *

PATHWAY OF TRYPAN BLUE FROM PARANASAL SINUSES TO CHEST BY LYMPHATIC AND BLOOD STREAM ROUTE

1. Sinus lymph spaces and small lymphatics.
2. Retropharyngeal and deep superior cervical lymph nodes.
3. Cervical lymphatics.
4. Subclavian vein, superior vena cava, right heart.
5. Pulmonary capillary bed.
6. Diffusion through capillary endothelium.

7. Phagocytosis by septal cells, some of which become detached as alveolar "dust cells."
8. Migration of septal cells into perivascular and peribronchial lymphatics, in interior of lung, and into pleural lymphatics from outer portion of lung.
9. Drainage of perivascular, bronchial and pleural lymphatics into bronchial and mediastinal lymph nodes. Enlargement of lymph follicles along intrapulmonary bronchial walls.
10. Alveolar "dust cells" discharged with mucus through bronchial passages.

* * *

PATHWAY OF TRYPAN BLUE FROM POSTERIOR NASOPHARYNX BY TRACHEAL ROUTE

1. Trachea, bronchi, bronchioles.
2. Air-sacs.
3. Phagocytosis by septal cells and alveolar "dust cells."
4. Trypan blue laden septal cells migrate into peribronchial and perivascular lymphatics and bronchial lymph follicles. Some migratory cells in pleural lymphatics.
5. Drainage into bronchial and mediastinal lymph nodes.

* * *

Since the above described experiments were completed, my attention has been directed to the evidence presented by Ravenel and Reichel[18] that tubercle bacilli may pass through the intestinal wall and reach the lungs by way of the thoracic duct and right heart. Although it has been repeatedly demonstrated that epithelium is very resistant to trypan blue and related substances, the fact that our animals ingested through the œsophagus considerable amounts of the dye during the course of an experiment made it appear desirable to examine this possible pathway of entry into the lungs. Accordingly, a kitten was given 30 cc. of 0.5 per cent solution of trypan blue, by mouth, daily for five days. The solution was thoroughly mixed with the ordinary animal-house ration so that virtually all of it, a total of 150 cc., was consumed. This kitten (number 22) was about the same age and size as number 21, which had been injected intratracheally with a total of about 7 cc. trypan blue solution also during a period of five days.

Kitten number 22 passed blue fæces from the first day of the experiment. It was killed on the fifth day, two hours after the last meal containing dye mixed with the food. Digestion was in full progress. When the abdomen was opened there was no evidence of coloration by the dye, either of the wall of the digestive tube or of the lacteals issuing from it. The latter were plainly visible as white threads in the mesentery. The digestive tube itself, when opened, revealed a continuous mass of deep blue colored food and fæcal matter throughout its length. Sections of œsophagus, stomach, duodenum, jejunum, ileum, and colon, stained with safranin, gave no sign of absorption of trypan blue by these organs. Sections of lungs, liver, spleen and kidney likewise were free of trypan blue granules. The intestinal route of absorption can therefore be ruled out in our experiments.

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Instructional Subjects

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One Panel Discussion
Three Special Courses — A, B, and C
One Special Exhibit
Individual Courses
138 Ophthalmological periods — 49 Instructors
122 Otolaryngological periods — 43 Instructors

* * *

Panel Discussion

LYMPHATIC DRAINAGE OF THE HEAD

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SPECIAL COURSES

A. HISTOPATHOLOGY OF THE EYE

Dr. Milton L. Berliner, New York
Dr. W. E. Camp, Minneapolis
Dr. T. L. Terry, Boston
Dr. Georgiana Dvorak-Theobald, Oak Park, Ill.

B. HISTOPATHOLOGY OF THE EAR, NOSE AND THROAT

Dr. Joseph C. Beck, Chicago
Dr. LeRoy A. Schall, Boston
Dr. Gregor McGregor, Toronto
Dr. Frank Novak, Jr., Chicago
Dr. Harris P. Mosher, Boston, *Consultant*

C. ANATOMY OF THE HEAD AND NECK

Dr. Oscar V. Batson, Philadelphia

* * *

CONFERENCES

OPHTHALMOLOGY

DR. FRANCIS HEED ADLER, Philadelphia
The Pupil in Health and in Disease

DR. THOMAS D. ALLEN, Chicago
Summary of Modern Opinions Upon Cause of Retinal Detachment

DR. WALTER S. ATKINSON, Watertown, N. Y.
Local Anesthesia in Ophthalmic Surgery

DR. CHARLES A. BAHN, New Orleans
Ophthalmic Endocrinology

DR. OTTO BARKAN, San Francisco
Chronic Glaucoma

DR. ARTHUR J. BEDELL, Albany
The Value of Photography in Ophthalmology

DR. M. N. BEIGELMAN, Los Angeles
Vitreous: Anatomical and Clinical Considerations

DR. WILLIAM L. BENEDICT, Rochester, Minn.
Orbital Tumors

DR. CONRAD BERENS, New York
The Presbyopic Patient

DR. ALBERT L. BROWN, Cincinnati
The Search of Etiology of Uveitis

DR. GORDON M. BRUCE, New York
Contact Glasses: Theory, Clinical Use, Demonstration

DR. RAMON CASTROVIEJO, New York
Modern Aspects of Corneal Transplantation

DR. C. A. CLAPP, Baltimore
Practical Points in Cataract Surgery

DR. GRADY E. CLAY, Atlanta
Medical Ophthalmology

DR. MARTIN COHEN, New York
Treatment of Trachoma

DR. FREDERICK C. CORDES, San Francisco
Early Simple Glaucoma

DR. WILLIAM H. CRISP, Denver
Pointers in Examining and Prescribing for Hyperphoria, Myopia, Astigmatism, Presbyopia

DR. WILLIAM THORNWALL DAVIS, Washington, D. C.
Fusion Treatment of the Phorias

DR. E. C. ELLETT, Memphis
Use of Sutures in Cataract Surgery

DR. JOHN N. EVANS, Brooklyn
The Scotomas of Glaucoma

DR. LOUISE FARWELL, Chicago
Psychology of Vision

DR. F. BRUCE FRALICK, Ann Arbor
Retro-bulbar Neuritis

DR. JONAS FRIEDENWALD, Baltimore
Circulation of the Intra-ocular Fluid

DR. RICHARD C. GAMBLE, Chicago
Orbital Abscess

DR. HARRY S. GRADLE, Chicago
The Congestive Forms of Glaucoma

DR. LEGRAND H. HARDY, New York
Orthoptic Training

DR. H. ROMMEL HILDRETH, St. Louis
Nerve-block Anesthesia for Ophthalmic Operations

DR. P. CHALMERS JAMESON, Brooklyn
The Recession Operation

DR. HAROLD H. JOY, Syracuse, N. Y.
Sympathetic Ophthalmia

DR. BEN WITT KEY, New York
The Refinements of Refraction

DR. DANIEL B. KIRBY and
DR. J. P. MACNIE, New York
Indications For and Type of Operation Best Used in Detachment of the Retina

DR. WALTER B. LANCASTER, Boston
The Physiology of the Motor Anomalies of the Eye

DR. ALBERT N. LEMOINE, Kansas City, Mo.
Treatment of Tuberculous Conditions of the Eye

DR. WALTER I. LILLIE, Philadelphia
The Diagnostic Value of Anomalies of the Visual Fields

DR. JOHN S. LUNDY, Rochester, Minn.
General Anesthesia in Eye, Ear, Nose, and Throat Operations

DR. ROBERT J. MASTERS, Indianapolis
Recent Advances in Slit-Lamp Microscopy

DR. SIDNEY L. OLSHO, Philadelphia
The Prescribing of Glasses

DR. AVERY DE H. PRANGEN, Rochester, Minn.
Practical Points in Refraction

DR. A. D. RUEDEMANN, Cleveland
Ocular Allergy

DR. BERNARD SAMUELS and
EDGAR B. BURCHELL, New York
The Morbid Anatomy of the Ciliary Body

DR. HUNTER SCARLETT, Philadelphia
Medical Ophthalmology

DR. E. B. SPAETH, Philadelphia
Plastic Surgery About the Eye

MAJOR FREDERIC H. THORNE, Washington, D. C.
Ophthalmology and Otolaryngology in Aviation

DR. PHILLIPS THYGESON, New York
The Treatment of Conjunctivitis

DR. FREDERICK T. TOOKE, Montreal
Ocular Tuberculosis

DR. CLIFFORD B. WALKER, Los Angeles
Separated Retina, Pathology and Treatment

DR. E. L. WHITNEY, Detroit
The Medical and Surgical Treatment of Tear Sac Disease

DR. MEYER WIENER, St. Louis
Important Points of Technic in Eye Surgery

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OTOLARYNGOLOGY

DR. JAMES A. BABBITT, Philadelphia
Tinnitus Aurium and Vertigo

DR. JOHN F. BARNHILL, Indianapolis
Anatomy and Associated Phenomena of Larynx

DR. JOSEPH C. BECK, Chicago
Treatment of Malignancies of the Nose and Sinuses

DR. LOUIS J. BIRSNER, St. Louis
Applied Surgical Anatomy of the Paranasal Sinuses

DR. HARRY P. CAHILL, Boston
The Diagnosis of Intracranial Lesions

DR. THOMAS E. CARMODY, Denver
Examination of the Oral Cavity by Sight and Palpation

DR. LOUIS H. CLERF, Philadelphia
Differential Diagnosis of Hoarseness

DR. ALFRED J. CONE, St. Louis
Physical Agencies in Otolaryngology

DR. KENNETH M. DAY, Pittsburgh
Chronic Suppurative Otitis Media

DR. LEE W. DEAN, St. Louis
Pediatric Otolaryngology

DR. WELLS P. EAGLETON, Newark
Intracranial Complications—Especially in Relation to Apex Infections

DR. RALPH A. FENTON, Portland, Ore.
Ocular Disturbances of Nasal Origin

DR. A. C. FURSTENBERG, Ann Arbor
Clinical and Pathological Study of Tumors and Cysts of the Nose, Pharynx, Mouth and Neck of Teratological Origin

DR. THOMAS R. GITTINS, Sioux City, Iowa
Headache: Differential Diagnosis

DR. PERRY G. GOLDSMITH, Toronto
Failures and Errors in the Practice of Otolaryngology

DR. W. E. GROVE, Milwaukee
Ear and Head Injuries

DR. L. K. GUGGENHEIM, St. Louis
The Etiology, Pathogenesis, Histopathology and Treatment of Otosclerosis

DR. FRENCH K. HANSEL, St. Louis
Allergy of the Nose and Paranasal Sinuses

DR. FRED Z. HAVENS, Rochester, Minn.
Fractures of the Bones of the Face

DR. SAMUEL IGLAUER, Cincinnati
Deep Suppuration of the Neck

DR. CHEVALIER JACKSON and
DR. C. L. JACKSON, Philadelphia
Cancer of the Larynx

DR. V. H. KAZANJIAN, Boston
Plastic Surgery of the Face

DR. SAMUEL J. KOPETZKY, New York
Present Conception of Otitic Meningitis

DR. OLOF LARSELL, Portland, Ore.
The Sympathetic System in Relation to Otolaryngology

DR. FREDERICK M. LAW, New York
X-Ray Interpretation of Sinuses and Mastoids

DR. FRANCIS E. LEJEUNE, New Orleans
Direct and Indirect Laryngoscopy

DR. HAROLD I. LILLIE, Rochester, Minn.
Physiology of the Nose

DR. ROMEO A. LUONGO, Philadelphia
The Fronto-Ethmo-Sphenoidal Operation

DR. M. H. LURIE, Boston
Deafness Explained by Modern Physiological Research

DR. WILLIAM J. McNALLY, Montreal
Anatomy and Physiology of the Internal Ear as a Basis for the Interpretation of Clinical Tests

DR. J. L. MAYBAUM, New York
Labyrinthine Tests in Intracranial Lesions

DR. WILLIAM MITHOEFER, Cincinnati
Treatment of Nasal Accessory Sinus Disease

DR. HARRIS P. MOSHER, Boston
The Esophagus

DR. FRANK J. NOVAK, JR., Chicago
Submucous Resection of the Septum

DR. SAMUEL J. PEARLMAN, Chicago
Differential Diagnosis of Deafness

DR. A. G. POHLMAN, Omaha
Auditory Test Methods from the Viewpoint of Practicability in Diagnosis

DR. EDGAR E. POOS, Detroit
Paralysis and Neuroses of the Larynx

DR. ROBERT F. RIDPATH, Philadelphia
The Sphenoid Sinus

DR. JOHN J. SHEA, Memphis
Control of Hemorrhages

DR. E. A. SPIEGEL, Philadelphia
Intracranial Investigation of the Vestibular Nerves

DR. THOMAS G. TICKLE, New York
Operative Procedure and Treatment of Facial Paralysis Due to Injury or a Bell's Palsy

DR. GABRIEL TUCKER, Philadelphia
Tracheotomy

Motion Pictures
and
New Instruments

The American Academy
of Ophthalmology and Otolaryngology
Forty-First Annual Meeting
New York, 1936

OPHTHALMOLOGY

* * *

THE SAFAR MULTIPLE DIATHERMY PUNCTURE OPERATION FOR RETINAL DETACHMENT

D. K. Pischel, M.D.

SAN FRANCISCO

The operation which I intend to show is the multiple diathermy puncture operation of Safar developed by him in 1932. This operation endeavors to cure detachments of the retina according to Lindner's theory of sealing off the affected area from the remainder of the fundus, rather than just trying to close the tear or hole.

In this operation the line of fine pins introduced while the diathermy current is flowing through them causes a solid line of exudative choroiditis to develop. This exudate will seal the retina firmly to the choroid. The retina comes into contact with it as the subretinal fluid drains out through the many pin holes in the sclera. Thus the affected area of the retina is surrounded by a solid wall of chorio-retinal adhesions and the detachment which always starts with the hole or tear cannot spread to the remainder of the fundus.

Since we know the retina is firmly attached at the ora serrata, we use this as part of the line of adhesion in case the tear is near it. In the Safar operation the diathermy punctures are made with many small pins which are left in place after being inserted in the sclera. This is done to prevent any premature escape of subretinal fluid, thus preventing the eyeball from becoming soft. It also allows the retina to be kept at a safe distance from the choroid where it cannot be traumatized. When the pins are removed the holes allow adequate drainage for the subretinal fluid.

The operation I am going to show was done in a case of a disinsertion at the ora serrata. This case was one of 18 months' duration, but I am glad to say was brought to successful conclu-

sion with final vision of 20/40, although the initial vision had been ability to count fingers at 1 meter.

The strength of current used for inserting the pins was 60 M.A. Any diathermy machine with a very sensitive ammeter and high frequency can be used.

The forceps for holding the pins can be obtained from V. Mueller & Company, Chicago. The pins are made by the U. S. Optical Company of Los Angeles.

THE ORTHOPTIC TREATMENT OF SQUINT*

WILLIAM THORNWALL DAVIS, M.D.

WASHINGTON, D. C.

IT WILL BE attempted by means of a short moving picture to show the salient points in the orthoptic treatment of squint, demonstrating the instruments used, the diagnosis of abnormal retinal correspondence and emphasizing the measurement of squint in full accommodation.

Satisfactory orthoptic work can only be performed by use of a synoptophore, or an instrument of that type. Patients are first placed before the synoptophore for measurement of squint, diagnosis of normal or abnormal retinal correspondence to ascertain if there is simultaneous macular perception, and the degree of fusion, if present. If normal retinal correspondence exists, i.e., if the image in each eye falls upon the macula, thereby resulting in true projection, ocular exercises may be prescribed with safety on other types of fusion instruments where the eyes cannot be watched. If abnormal retinal correspondence is present, i.e., the image does not fall upon the macula of the deviating eye, but at an eccentric point, false projection will be present. The synoptophore is used until this condition no longer exists and the brain has been stimulated to send its reflexes to the heretofore unused macula. Abnormal retinal correspondence is diagnosed on the synoptophore in the following manner. Two simultaneous macular perception charts are placed in the instrument, one before each eye. If the patient can superimpose these charts, fusion charts may also be used for making the test. The patient is asked to move the arms of the synoptophore until the two charts are superimposed or fused. For example, if dissimilar charts like the rooster and the cage, illustrated in the film, are used, the rooster must be placed in the cage. If fusion charts are used the patient attempts to blend

* From the Department of Ophthalmology, George Washington University School of Medicine.

the two. The angle at which the instrument is set is then read from the scale. If the subject has a convergent squint, for example, and has abnormal retinal correspondence, the reading will be lower than his actual angle of squint; it may be even on the divergent side of the scale.

While the patient is attempting to fix upon a central object, the two lights before the eyes are alternately lighted and extinguished. If his correspondence is abnormal, two things will be noted. First, when the lights are alternately flashed on and then off, the deviating eye which has been using an eccentric point on the retina will shift so that the image falls upon the macula. Secondly, the corneal reflex from the deviating eye will not be in the center of the cornea. The angle kappa must be taken into consideration. When the light is off before the fixing eye the squinting eye will turn out to fix in a case of convergent squint and will turn in to fix in a case of divergent squint. When the light is extinguished before the squinting eye it resumes eccentric fixation. This shifting of the eyes in abnormal retinal correspondence we have tried to show in the moving picture.

The angle kappa should be measured in all cases of squint both on the perimeter and on the synoptophore. It is possible to calibrate it in prism dioptres on the synoptophore. Patients having a wide angle kappa are not confused with those having abnormal retinal correspondence because the corneal reflexes are not centered.

It is also important to differentiate between the shift due to the breaking up of fusion and accommodation and that of abnormal retinal correspondence; otherwise, an improper diagnosis might be made. Both in divergent and convergent squint the patient may accommodate and manifest excessive convergence as he fuses. If the retinal correspondence is normal it will be noted that the corneal reflexes are centered. The eyes are at first still and the corneal reflexes in normal position. After the lights have been alternately flashed on and off before the eyes a few times, shifting may be noted which was not present when the lights were first flashed. To correct for movement of the eyes in a case of divergent squint, the arms of the synoptophore should be moved toward the divergent side a dioptre or two at a time; for convergent squint, the arms of the instrument are moved towards the convergent side. The lights are alternately flashed at intervals of two or three prism

dioptres. Each time this is done the position of the corneal reflex of the squinting eye will be noted and the adjustment of the arms of the synoptophore continued until the eye does not have to turn in order to attain macular fixation. The flashing of the lights is continued until the place is found on the scale where there is no shifting. This method breaks up the accommodation in the same way that the cover test does, so that true measurement of the squint is obtained.

After the above tests are made, the squint is measured by the corneal reflexes, setting the arms of the synoptophore so that the light from each tube shines on the center of the cornea. The amount of squint in prism dioptres or in degrees of arc can be read from the scale, allowing for the angle kappa.

To overcome false projection and eccentric fixation the instrument is locked at the true angle of squint and oscillated back and forth. The image before the squinting eye is therefore on the macula and if he is to see the picture before that eye the patient must use his macula. Mental effort is stimulated by the technician until the two pictures can be fused at this angle. Until the patient can accomplish this no other form of exercise is given. False projection must be completely eliminated before further steps can be pursued. Thus, it will be noted that before home exercises with the stereoscope can be given, it is necessary that the abnormal retinal correspondence be corrected. If this is not done the false projection may become fixed and the opportunity for the cure of the squint will be lost.

Emphasis is placed on the desire to cure the squint without glasses, if this be possible.

We have found that 36 per cent of the patients with squint who came to the clinic have abnormal retinal correspondence. In private work we saw only 17 per cent with abnormal retinal correspondence. This difference is probably due to the fact that the clinic patient is not seen for treatment until the school age, whereas in private work the squinting child is more likely to appear for treatment as soon as the squint manifests itself. We have, therefore, a lower incidence of amblyopia and false projection in the private patients.

It is difficult to obtain accurate visual acuity readings in young children. It is essential that the child be made comfortable and happy during this examination; otherwise, he will cooperate up to a certain point, then being tired of the whole procedure, state that

he "cannot see any more." Children are prone to memorize charts very quickly. To overcome this particular factor the Isolated E is used. The E's from a Snellen chart are cut out and a size corresponding to each line is pasted on a round white disc of cardboard. As the disc is round, it can be rotated in any direction and the child cannot memorize the direction. The usual letter, number and picture charts are used in preference to the Isolated E. We emphasize its use only in cases suspected of memorizing the charts.

All types of occlusion have been tried in the office and in the clinic but only by total adhesive occlusion have we had results that are worth while.

All cases should be measured in the nine cardinal fields, as stressed by Dr. James Watson White of New York, for an intelligent interpretation of the case. The card he uses for recording the findings is shown in the moving picture. Such measurements are made with the cover test and prisms and can be used successfully in a short time with young children. Before the diagnosis is assuredly made this test should be done, since it brings out vertical deviations. Surgery should not be done until the vertical deviation is studied. Where there is a vertical deviation combined with a divergent or convergent squint, if the lateral recti are operated upon, a wide swing in the opposite direction may occur. The vertical deviation, in such a case, is probably the cause of the lateral squint and since binocular vision was impossible, squint occurred as a defense reaction to overcome the diplopia and to aid in suppression.

It is important that all cases be measured by the prism and cover method with and without glasses for *distance and near and while fully accommodating* upon a picture or chart that the child is *really interested* in seeing, *in order to keep the accommodation at maximum*. As soon as the child loses interest in the picture he is fixing upon, the accommodation relaxes. It is necessary, therefore, to keep him interested while the squint is being measured. If the accommodation is not so stimulated the patient is likely to manifest no squint or only part of his actual squint. I am quite convinced that many cases are measured and pronounced cured because they have been measured without having the accommodation stimulated. Those with accommodative convergent squint may learn to inhibit their accommodation and to adapt themselves to blurred vision. When they wish to see objects distinctly they accommodate and at the same time converge and the squint appears. This is demon-

strated in the moving picture by a boy who has had an accommodative squint for five years and has learned to accommodate at will. You will notice him with his eyes straight since the visual act is unaccompanied by accommodation or convergence as he looks at the chart at 20 feet. The visual acuity of this boy without glasses is 10/200 in each eye, tested separately. When he is not accommodating and therefore not seeing clearly, his eyes are straight, but he only reads 14/200 with both eyes at the same time; when accommodating he reads 20/70-i with both eyes and at the same time squints. The same condition obtains with his glasses, the angle of the squint being less when he is accommodating. We have seen a number of similar cases. This case well illustrates our belief that many cases of squint are thought to be cured when as a matter of fact they have been taught to voluntarily control the accommodation. Hence, *they squint only when accommodating.* Obviously, unless the measurements are made with the patient accommodating, no squint will be manifest in cases of this type. Such a patient is seeing with reduced vision and not using his accommodation.

Our evaluation of orthoptics as at present used is that it is a valuable and essential aid in the cure of squint. It has approximately the same value as refraction in this regard. It is not a cure for squint, but squint cannot be cured without it. The following cases from which these statistics have been compiled were most carefully studied. The evaluation of them was made without bias. We believe they give the true value of the orthoptic treatment of squint.

Statistics

From the Orthoptic Clinic of the Episcopal Eye, Ear, Throat Hospital, Washington, D. C., March 1935 to June 1936:

Total number of cases 112.

41 per cent or 46 cases had amblyopia. Of these, 37 per cent or 17 cases were improved two or more lines.

36 per cent or 40 cases had Abnormal Retinal Correspondence. Of these, 25 per cent or 10 cases overcame it.

There were 4 improvements in the angle of squint in 112 cases. (Improvement from 5 to 15 prism dioptres, 3.4 per cent).

There was 1 cure: a convergent squint of 40 prism dioptres reduced to a phoria.

Private Orthoptic Work from January 1935 to September 1936: Total number of cases 46.

35 per cent or 16 cases had amblyopia. Of these, 81 per cent or 13 cases improved two or more lines.

17 per cent or 8 cases had Abnormal Retinal Correspondence. Of these, 38 per cent or 3 cases overcame it.

There were 6 improvements in the angle of squint in 46 cases. (Improvement from 5 to 15 prism dioptres).

There were 2 cures: 1 convergent squint of 30 prism dioptres reduced to a phoria; and 1 divergent squint of 40 prism dioptres reduced to a phoria.

MEDICAL ILLUSTRATIONS FROM ANCIENT TIMES TO THE PRESENT

HARRY VANDERBILT WURDEMANN, M.D., Sc.D., F.A.C.S.

SEATTLE

MEDICAL ILLUSTRATION goes back to the paintings in the caves of the Cro-Magnons in which are depicted wounds and death, and are even perpetuated in the statue of the adipose cavewoman. There is something to be found in the papyri of the Egyptians, pictured by the priests, who not only took care of the souls of their subjects but also attended to their bodily miseries. A few medical hand-drawn pictures are yet extant from the Dark Ages. Three thousand years ago, a Chinese found that a carved piece of wood, wet with paint, could be impressed upon paper and a number of copies made, and relics of that ancient Kingdom of Cathay are preserved by some of these woodcuts. After a long interval of time, woodcutting was rediscovered in Europe in the second half of the fifteenth century.

For the purpose of medical illustration, artists were hired by physicians and had to practice in secret, making hasty sketches for their general information or as preparation for projected works of art. The dissection of the human body, or even many operations, were at that time not only against the law and subject to punishment even up to death by the civil authorities but also condemnation to Hell by the clergy. Among the artists who made medical and anatomical illustrations were da Vinci, Buonarroti and Raphael. In the latter part of the fifteenth century, by the discovery of anatomy, a quality of drawing not schematic but from nature led to the elimination of traditional mistakes, to the increase of anatomic knowledge, and improvement of medical illustration. About this time, copperplate etching or tooling was evolved, and from then on, some of the most beautiful and correct anatomical illustrations were made, of which some copies persist to the present day. Vesalius, in 1543, attained all three ends at once in the most striking manner, and enriched the study of anatomy in the beautiful artistic mode of representation. From 1627 to 1737, the art of

copper engraving was perfected and a demand then came for pictorial anatomy; Bauhein and Casserior responded, Albinus and von Haller. Perhaps the first surgeon who was famous as a graphic artist was Peter Camper, who executed his own drawings. Among the early illustrations of pathologic subjects are those by William Hunter on the pregnant uterus, and Cheselden who employed the foremost artists of England in their execution. We might go on indefinitely with the older anatomists and artists, some of whose copper plates were colored by hand. Very few anatomists were themselves artists, but among those in the last century were Wilhelm His, Joseph Leidz, and Joseph Lister.

The day of the massive, expensive atlases of wood, copper, steel and mezzotint engravings is over, and we now have the photographic processes which are well illustrated in many of our magazines. Particularly in the line of our specialties are the illustrations of Frost, Haab, Oeller, Jaeger, Liebreich, Ramsay and Oatman, who made their own drawings; of Schieck and Wilmer and Casey A. Wood for the eye, who employed others; Türck, Politzer, Schnitzler, who employed others as artists for the nose and throat.

While any kind of copy from the most crude drawings to finished paintings, photographs, or even objects, may be reproduced by the photographic art and its child, photo-engraving, the better the copy, the better the result when printed. For our purpose, pencil, pen and ink, monochrome brush work, photographs, watercolor or oil paintings make good copy. This is usually entrusted by the physician wanting illustrations for his article or his book to a hired artist. A few medical men are artists of this kind and from these lasting representations of their cases are perpetuated in the few atlases published, Margaretta Washington, Annette Burgess, Beard, Oatman, Feinberg, and myself being among those from America. Most of the others for eye, ear, nose and throat work have emanated from Germany or Austria. However, there are a number of good special artists in Great Britain and France, as Arthur Head; but few of them are medical men. In those atlases published abroad, I have contributed, myself, to the recent edition of Schieck's *Handuch f. Augenheilkunde* and, years ago, to Schnitzler's *Laryngoscopic Atlas* and Politzer's *Ohren-bilder*. The exhibit at this session embraces samples of ophthalmic pictures for a proposed *American Atlas of Ophthalmology*, unfortunately an expensive undertaking needing the backing of the medical profession to insure the interest of a publisher.

It goes without saying that no matter how skilled the artist, how faithful to the reproduction of the object as seen by him in drawing and in color knowledge, unless he is a skilled medical man and particularly a specialist in his field, it is impossible to fully tell the tale by a picture that is done secondhand.

The soul of the subject can not be mechanically represented and reproduced in a photograph as well as it can be by the artist who knows his subject, not only pictorially but professionally. No photograph of a human could ever reproduce the smile of the Mona Lisa as represented by da Vinci, a smile that will last through the ages and is a composite of the emotions. Therefore, no matter how well developed the art of photography becomes, a drawing made by a medical man, whether crude or finished, tells the tale in a manner that no hired hand can accomplish. The lesson to be taught by a picture is not that of a view, a fleeting glimpse of one phase; but particularly in medical representation, the diagnosis must be accentuated by a little overdrawing in light, shade, and color, and the other parts of the picture of no import be kept somewhat in the background.

In recent years most excellent photographs of the eye have been made, especially those by Dr. Bedell of the fundus, and Dr. Bane of external disease, who lately has taken up color photography with some success.

Of all the anatomic woodcuts, we always return to those of Gray's *Anatomy;* the drawings made by Dr. H. V. Carter in 1850 and reproduced in the many editions of the famous work.

A little bit of personal history comes here. In 1888, at the time of the author's graduation, he was working with Professor Elliott Coues of the Smithsonian in the preparation of a new anatomy and we had a contract with a well-known publishing house for the manuscript, with one thousand illustrations. The author had colored his own copy of Gray's *Anatomy*, the veins in blue, arteries in red, nerves in yellow, for his guidance, and showed this to Professor Coues, who took it on to the publisher; and a few weeks later, our contracts were abrogated for the reason that they "were bringing out a new edition of Gray's *Anatomy*" — and, lo and behold! This came out in color. Enough said!

"There are tricks in all trades but ours." And there are many in the illustrative art. The crudest drawing may be furbished up to look like the work of an artist.

I have found many time-saving shortcuts. For instance, a dimly printed photographic print may be used as a background of an

exact drawing, the detail being brought out by pencil and brush. Line drawing is most difficult. Lettering is accomplished by but few. Here, black ink, good paper, and sharp lines are essential. These may be reproduced by zinc etching, the cheapest process. Wash drawings and, above all, colored pictures, are more difficult, but even here the background of light and shade may be made by pencil, dark ink and brush, over which may be laid the color. For ophthalmoscopic pictures, a red cardboard may be the foundation, saving much time in getting the tint of the fundus. The best water-colors and finest sable brushes should be used; even the tyro may make a passable painting by these methods.

Reproduction for illustrative purposes of original drawings, paintings and photographs is the highest attainment of this form of illustrative art. It dates back to the early part of the nineteenth century by the discovery of Daguerre that an image formed by an optical lens could be permanently fixed upon a copper or glass plate by the use of silver compounds. Then reproductions by photography were printed in black and white or monochrome; colors being added by the artist to each print afterwards, as in tinting photographs to a similitude of nature.

As early as 1869, Hauron evolved a heliochrome process in which three negatives were printed on bichromatized gelatin films which were stained yellow, red and blue, and superimposed to produce the color effect. Later on was evolved photo-lithography in black and white and then in color, by which many well-known reproductions of paintings and of anatomic and pathologic atlases were made. This was done mostly in Europe, the Germans, particularly, excelling.

Later on, in America, it was discovered that by superimposing a screen of finely-ruled lines, crossing each other at right angles behind the photographic lens, a negative could be obtained which was composed of square dots. This, when used to print on a copper or zinc plate sensitized by a chemical film of albumen, impregnated with silver, and then immersed in an acid bath with the acid eating the spots not touched by light, obtains a relief plate from which, when inked, many impressions are made.

Later on, and really only recently, light filters were used in the camera; i.e., of orange, green, and violet, which eliminate in each plate all the rays of light except the one desired, and then the three plates are printed with suitable inks. These are not the same color as the filters. The pigments used are yellow, blue, and red. A fair reproduction in color of the object photographed can be printed

from these plates. This is the three-color process. In addition to this, however, a black plate is also used. This accentuates the light and shade, and, when the four are printed, makes the four-color process.

The halftone, or black and white, has been used commercially for about 37 years, the color process engraving for about 30 years. Previously to this, a number of methods such as photo-lithography and offset printing, the latter of which is by slightly "fudging" the different superimposed colors, were used; but while these latter have a special sphere for reproductive fidelity and sharpness of detail, the relief photo-engraving processes surpass all.

We have recently had the development of color photography by which a photographic print may be made by one exposure and in one printing. This is an expensive process for illustrations and does not allow of the handiwork of the photo-engraver artist to bring out the salient points of the picture and to subdue those that are of no interest and which do not, from either an illustrative, educational, or artistic standpoint, show the story. Technically, the reproduction of a drawing, photograph, or painting, the mechanical part of the work, as well as the printing and the paper, must be correlated to the purpose, whether it be from the coarse paper and rotary press of the newspaper, to the finest kind of super-calendered paper for book work.

The line screens vary from 50 lines to the inch to 175 lines to the inch; the latter bringing out full detail but being very difficult to print. The average halftone screen is from 100 to 120 lines an inch. It was found that in process color work, the screen angles of the filter should not be at 45° and 135°, at which angles diagonal to the square of the plate, the lines are least perceptible to the eye, as in monochrome reproduction. The best effects are here obtained by shifting the angle to 45° red, 105° blue, and in making these for the yellow color in the ultimate print. The violet filter was used for the red, the green for the blue, the orange, and for the black, the yellow.

Regardless of the most intelligent manipulation, the four negatives of a color set are seldom more than the crude foundation of the finished result which depends upon skill and talent due principally to the fact that no dyes have been discovered that will make perfect color filters or render silver salts completely sensitive to all colors; the color separations are not exact. Consequently, the task of correcting these differences falls to the etcher, a task which, in addition to mechanical skill and artistic technique, requires a

thorough knowledge of color composition and consumes most of the time required to produce color plates. As a consequence of this, there are exactly 98 handlings in the production of plates alone for a book illustration in color before it goes to the printer's hands. Here again comes a labor that can only be entrusted to an artist in the selection of the proper inks, the overlaying and underlaying, the proper kind of paper, the holding of the prints themselves, altogether, perhaps 100 different procedures.

Going back of this is the copy, whether made by the artist in oil or watercolor, or the photographer, and every one of which, from the artist to the bookbinder, must have a penchant and training as well as natural artistic ability to bring out the wonderful color illustrations that are now available.

It may be interesting to know that in the United States and Canada there are about 9000 photo-engravers, all of whom must serve six years as apprentices before they become journeymen; this is almost equivalent to the education required for medicine or law. However, in the three learned professions, the student pays his own way. As a rule, his time is so much taken up with his studies that he is unable to earn his keep during that time but here, in an artistic vocation, commonly classed as a "trade," while there are no ultimate highly-paid workers, yet the apprentice has the following scale:

First year $15; second year $18, third year $27, fourth year $37.50, fifth year $45, sixth year $53 a week. After that, some journeymen photo-engravers make as high as $300 a month. Naturally, the superintendents and managers of certain engraving companies are paid somewhat higher. The scale of wages certainly is as good, or better, than that of the average doctor or lawyer, and admits of a vocational life of at least twice that of these two learned professions, with just about as much emolument. The rather good pay reflects itself in the price of cuts.

Medical men who make sketch drawings for their case reports acquire a photographic memory, and those of us who have taken this up as a hobby get considerable personal pleasure, and are able to demonstrate cases and ideas more fully in lectures or in print. The Chinese say, "A picture gives as much information as 10,000 words," and nowadays we say, "A picture is worth a page of print."

A PHOROMETRIC STEREOSCOPE*

EMANUEL KRIMSKY, M.D.

BROOKLYN

A PHOROMETRIC or calibrated stereoscope enables the examiner to determine at a glance the amount of convergence or divergence, with selective accommodations, for fusing split pictures, by incorporating:

a. Viewing lenses of known focal length and variable separation.
b. Calibrated rod.
c. Viewing boxes calibrated to indicate the amount of separation of split pictures.
d. Table to which examiner may refer so as to readily translate these vergence readings in terms of prism ductions or phorias, thus enabling him to determine the vergence status in the primary phase both at infinity and at any desired accommodative range, to diagnose and to classify the type and degree of muscle anomaly, the range of fusion, the amount of phoria, and the progress in terms of prismatic vergence with each sitting.

The operation of a precision stereoscope is simple, and is based on an appreciation of a few underlying principles. These were presented as part of my scientific exhibit at the 1935 A.M.A. convention. In short, they may be summarized as follows:

a. The eyes cannot maintain the same pupillary separation with changes in viewing distance.
b. As the eyes converge with accommodation, the interstereogram separation must be made correspondingly less in order to maintain easy fusion.
c. A split stereogram which can be shifted to correspond to changing positions of the visual axes with variable accommodations is the only practical method for stereoscopic study.

* Made by the Precise Instrument Co., 200 Tillary St., Brooklyn, N. Y.

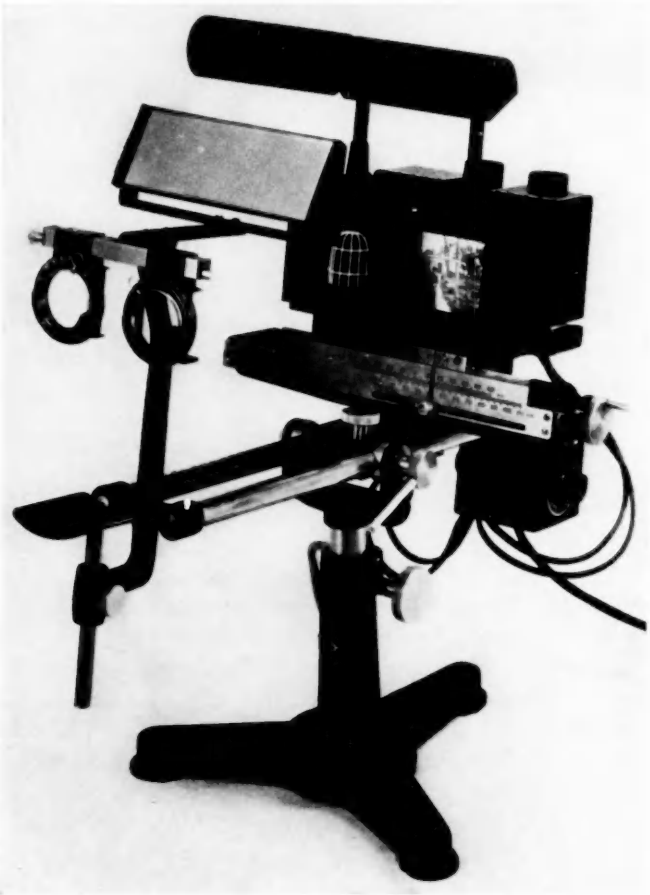

FIG. 1 — (Krimsky) — Author's Phorometric Stereoscope (septal curtains released from attachment to viewing frame).

d. A stereogram with separation fixed for infinity viewing range becomes relatively divergent when brought nearer to the eyes.
e. The variable convergences of the eyes with changing accommodations may be accurately calculated and applied to stereoscopic investigation.

From a mechanical standpoint, my stereoscope presents the following features: Leaves the choice of viewing lenses and prisms to the practitioner; adjustable, calibrated lenticular separation; improved method for attaching one or more prisms; con-

necting rod unusually long; free from hindrances which limit the desired approximation of viewing head and viewing box for testing eyes in the convergence phase; ready adjustment of instrument in see-saw positions for testing eyes in upward or downward gaze; enables the examiner to readily shift to transparencies, opaque prints, or solid objects for training or examining material; accurate control of picture separations; no clumsy finger manipulations; novel device for observing position of patient's eyes as an aid to accuracy in investigation; flexible telescopic septum automatically adjustable to changing viewing distances; stereo-campimetric attachment so as to serve as a combination instrument; independent lighting units with shifting control for either transparencies or opaque prints, rheostat, flasher, etc.

With the use of suitable material, my instrument may be readily adapted to convergent squints amounting to at least 50 prism diopters without the use of supplementary prisms; and to divergent deviations of considerably greater amount.

A phorometric stereoscope has the following advantages over a synoptophore:

a. Examiner may determine the fusional status and fusional reserve at any desired accommodative range.

b. Accommodational convergence may be varied at will by merely changing the viewing distance; also, the infinity plane may be varied at will by merely inserting lenses of desired focal length.

c. Not only may transparencies be used, but also opaque paper prints and photographs, as well as solid objects. Moreover, by the use of adapters, different sizes of transparencies or opaque prints may be employed.

d. Offers the examiner a more ideal method for prescribing home training exercises with a companion stereoscope.

ASTIGMATIC DIALS IN REFINED REFRACTION

* * *

Suggestions for Experiments with Lancaster-Regan Astigmatic Dials to Show the Principles Involved

James Joseph Regan, M.D.

Boston

Those who are not skilled in the use of astigmatic dials will find the following experiments a valuable means of clarifying the principles involved.

How to Illuminate the Dials

Hang the charts in a good light of about 15 to 25 foot candles, flood-lighting the whole wall where they are hung so that the wall around the dials will be almost as bright as the dials themselves. Avoid a dark background for two reasons: first, it is desirable to have light equal to that found under average working conditions — neither the large pupil of dim illumination nor the small pupil of over-bright illumination is desirable. The larger pupil introduces into the problem the refractive errors outside the visual zone of the cornea. These are often substantial. The small pupil reduces the size of circles of diffusion in a way similar to a stenopaic disc, although of course to a less degree, thus obscuring the astigmatic effects. Secondly, the retina responds more sensitively (as shown by Cobb and others*) when the surrounding field is such that there is not a marked contrast in brightness between the chart and the surrounding field.

Try the following experiments on your own eye if you have one with approximately normal vision — 6/8 or better. If not, use an intelligent patient, preferably one with an accommodation of at least 4 D. and a pupil of 4 mm. or more.

Start with the eye corrected to approximate emmetropia.

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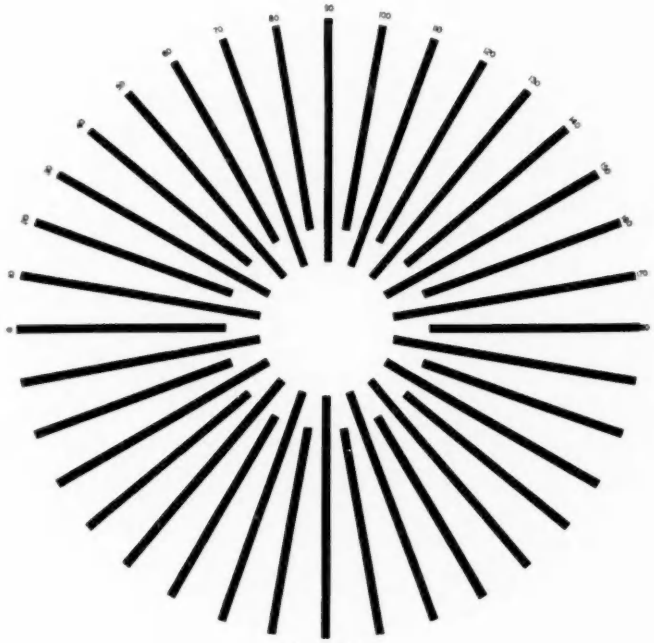
* Cobb: *The Effect on Foveal Vision of Bright Surroundings*, J. Exp. Psychol., January 1916, p. 540.
Cobb and Moss: *The Effect of Dark Surroundings on Vision*, Journal Franklin Institute, v. 206, p. 827, 1928.



Dial No. 1

Dial No. 2

EXPERIMENTS ON FOGGING

Place a trial frame comfortably over the glasses ordinarily worn. Occlude the eye not being tested with an opaque disc. Read the smallest letters possible on the chart at 6 meters.

(1) Place in the trial frame + 0.25, + 0.50, + 0.75 and + 1.00 spheres in succession, reading the smallest letters possible in each case and making a note of same. This will be referred to as "fogging."

(2) Do the same with — 0.25, — 0.50, — 0.75 and — 1.00 spheres.

(1) should show a reduction in vision definite for each addition of plus spheres. If no definite reduction occurs with + 0.50 it shows that there is some uncorrected hypermetropia. An emmetropic eye with a + 0.50 sphere reads about two lines less on a Green-Ewing chart.*

(2) should show no definite reduction in vision if the subject has good active accommodation.

(3) Repeat (1) with a series of opaque discs with apertures of different sizes ranging from 5 mm. to 2 mm. to reduce the size of the pupil. You should find that the fogging effect is markedly less with a small pupil.

(4) Repeat (1) with the other eye, recording findings. If the corrections worn by the two eyes are well balanced, the reductions in vision will be about the same with each eye.

(5) Repeat (1) with both eyes simultaneously. In the average case the reduction in vision will be markedly less than with one eye, showing that the subject relaxes his accommodation more when both eyes are tested together.

(6) Repeat (1) using stronger lenses up to 4 D. with the test object at 40 cm. = 2.50 D.

Repeat (2) at the same distance (40 cm.) using stronger concave lenses up to whatever power is needed to take up all the accommodation and produce some reduction in vision.

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* The great advantage of the Green-Ewing chart is that the test letters are graduated by geometric progression, making the ratio or difference in size the same with each successive line. For example: Letters on the 6/10 line are 25 per cent larger than the letters on the next line, and these in turn are 25 per cent larger than the letters on the next line, and so on for the whole scale. Distributed by C. V. Mosby Co., St. Louis.

EXPERIMENTS IN ASTIGMATISM

(7) Like (1) except that instead of observing the test letters, observe the astigmatic dial No. 1 (the one with 36 radiating lines at 10° intervals).

If the subject sees all the lines alike — equally distinct or, when fogged, equally blurred, either he has no marked regular astigmatism which is uncorrected by his glasses or, if he has some uncorrected astigmatism, he is not a keen enough observer to detect it.

(8) Place a plus sphere in the trial frame over one eye (the other being occluded with a blank disc), choose a plus sphere which will fog the eye to a vision of 6/10 or 6/8.

(a) Add a — 1.00 cylinder axis 90; observe and record the appearance of the astigmatic dial. The line at 90° and one or possibly two each side of it are much darker, more distinct, better focused than any of the others.

(b) Change the axis of the minus cylinder from 90° to various other axes and note how clearly lines in the chart corespond.

(c) Repeat (a) and (b) with — 0.75 cylinder, — 0.50, — 0.25 and — 0.12 cylinders.

(9) Repeat (8) with less fogging and without fogging.

(10) Repeat (8) with — 0.25 sphere, — 0.50 and — 0.75 spheres. Note that with the conditions as in (8), i.e., *fogged*, the chart shows the clear lines in the same axis as the minus cylinder, but under the conditions of (10) there may be no marked difference in the radiating lines with a cylinder which would be very easily detected if the subject were fogged; then as the minus sphere is increased, the time comes when the clearest lines are at *right angles* to the axis of the cylinder. With a little practice and by selecting the proper combinations of plus sphere and minus cylinder at different axes, the subject can make different axes come out clear at will, e.g., with minus cylinder axis 45° he can make the lines at 45° clear, or by changing his accommodation he can bring out the lines at 135°.

This shows how *worthless* the astigmatic charts are for finding the axis of astigmatism unless the accommodation is cared for by *fogging* properly.

Note also that with a small amount of astigmatism no difference in the radiating lines is detected if the fogging is *too* great. The ideal fogging is the sphere which fully corrects the hypermetropia plus another sphere which equals the amount of astig-

matism, e.g., if the true static refraction is + 1.00 sphere ⌒ + 0.75 cylinder axis 80°, the best fogging to show the astigmatism by the dials is obtained with a + 1.75 sphere.

Hence, when the *amount* of astigmatism has been determined in a given case, it is often wise to corroborate the axis by placing in the trial frame the sphere which equals the stronger axis in hypermetropia, and the weaker in myopia, and determine the axis of astigmatism again on the No. 1 dial.

(11) In a series of cases with astigmatism, either natural or produced by suitable sphero-cylindrical lenses, first determine the axis by the above method and then measure the amount by using dial No. 2, the one with the cross adjustable at any angle.

Set the cross at the angle determined by the test with the first dial. One line will be clearer than the other; this is the axis of the plus cylinder. The other line will be less clear; this is the axis for a minus cylinder to correct the astigmatism. Try minus cylinders at this axis until the one is found which makes the two lines alike (not the one which makes this line as distinct as possible, for this would over-correct the astigmatism). When the correct power is found the two lines of the cross will be equally distinct. (If a strong minus cylinder is required, more plus sphere must be added to maintain the *fogging*.)

This gives the first estimate of the *amount* of astigmatism present; that is, the strength of the cylinder required to make both meridians equal. To measure the spherical error more accurately (after which the astigmatism will be verified and a still more accurate measurement made), reduce the fogging until the maximum vision is found. Select a power such, that adding + 0.50 sphere reduces the vision from 6/5 to 6/8. Try the effect of a + 0.25, then a + 0.12 cylinder, with their axes first with the correcting cylinder, and then at right angles to the correcting cylinder. Unless the hypermetropia of the subject is not fully corrected, the subject should promptly and consistently tell which way you hold the cylinder. The cylinder held at either axis will blur that axis on the chart, thus making the opposite axis the more distinct one.

SOME DIFFICULTIES ENCOUNTERED

Subjects, *adequately fogged*, often view the No. 1 dial for several minutes and insist that all 36 lines look alike. If one holds a — 0.50 cylinder axis 90° before the eye, the lines may still appear alike. Hold the cylinder axis 180° before the eye, and the horizontal

lines may appear blackest. Direct attention to the No. 2 dial with lines vertical and horizontal, and the horizontal line appears blacker. Insert minus cylinder axis 90° until both lines appear equally black, and do not be surprised to find that frequently .50 D. or more astigmatism exists. Of course, when astigmatism is determined in this manner, great care must be exercised in determining the axis, and in such cases, we suggest trying the patient again on the No. 1 dial.

Rarely, no difference will be noted in the lines with a —0.50 cylinder in any position, and the response is that all the lines on the No. 1 dial look doubled or shadowed. If so, the subject may be over-fogged — an error which may occur when the astigmatism is small. Some individuals will select lines in two directions — not at right angles. In such cases the astigmatism is small and somewhat irregular. The subject is using a special part of the focal pencil which nowhere, in his case, brings the light to a perfect point focus. He chooses the place which gives him the best results in seeing.

The subject selects blackest lines on dial No. 1 and after setting the cross dial, minus cylinders up to —0.75 are inserted. At this point, the response might be — "Both lines appear double, but equally black." The most frequent cause of this is fogging. Therefore, make sure by holding a minus sphere before the eye. This usually removes this doubling. If not, try out the axis again on No. 1 dial. There are two ways of selecting the axis at this point. Reduce the sphere sufficiently to give 6/6 vision; then remove the minus cylinder from the frame and direct his attention to dial No. 1. The center of the group of black lines may now appear to be 5° or 10° either side of his original selection. Set the cross dial (No. 2) at the new axis. Increase the sphere to re-fog, insert the —0.75 cylinder and proceed toward accurate measurement of the amount of astigmatism. Another method is to reduce the sphere until the patient has 6/5 vision and rotate the cylinder with the patient viewing letter on the 6/8 or 6/6 line. The axis to choose is the one which gives the best focus of these letters.

CORNEAL CONTACT GLASS FOR EXAMINATION OF THE DEEP VITREOUS

HARVEY E. THORPE, M.D.

PITTSBURGH

THE CORNEAL contact glass* for examination of the deep vitreous which I am demonstrating is a modified form of the contact glass which Koeppe demonstrated in this country in 1925 for focal illumination of the retina and the study of the deep vitreous. Koeppe's

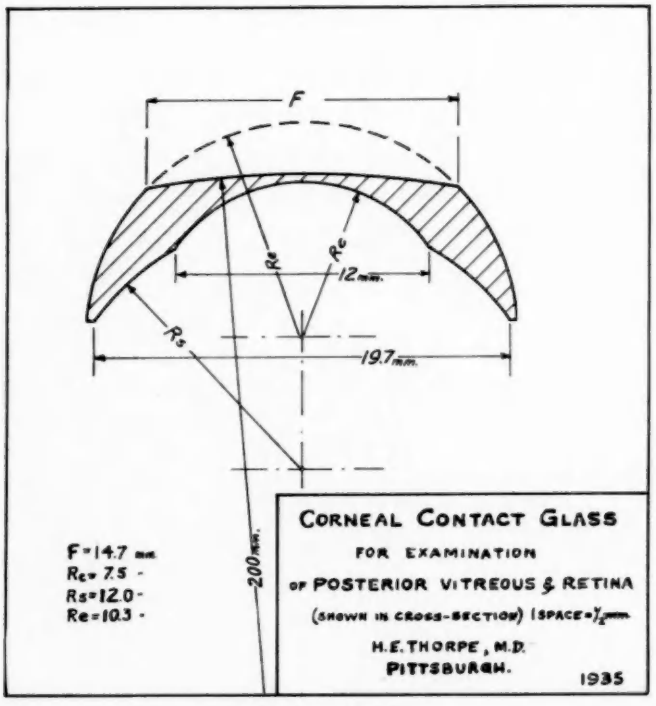

FIG. 1 — (Thorpe).

* This corneal contact glass was manufactured by Bausch & Lomb Optical Co., Rochester, N. Y.

lens has an anterior face of only 8 millimeters. This makes too narrow an aperture for entrance of the slit lamp beam and observation with the loupe or microscope.

Lindner had the contact glass modified so that it would have a diameter of 15 millimeters on its plano anterior face.

I have modified the anterior face of the above glass, giving it a convex surface. The lens is ground to have a scleral curve of 12 millimeters radius, a corneal curve of 7.5 millimeters radius, and a convex anterior face 14.7 millimeters in diameter, with a radius of curvature of 200 millimeters (see Fig. 1). It is thus possible to get a more magnified view of the deeper structures than with the contact glass modified by Lindner.

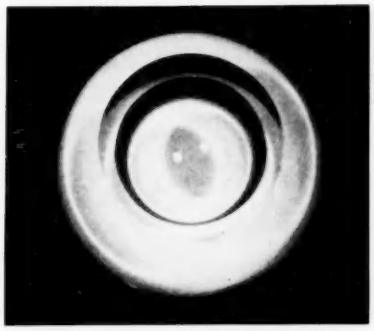

FIG. 2 — (Thorpe) — Contact glass for deep vitreous examination.

The pupil is dilated maximally and the contact glass filled with phyiological saline is introduced between the lids while the patient leans forward. It may be removed from the conjunctival sac by slipping a strabismus hook under its edge.

The deep vitreous illuminated by the focused slit lamp beam through the contact glass is covered with an ordinary loupe or with a monobjective microscope. It is possible to determine vitreous separation by this means. A beautiful miniature view of the fundus is also observed with the naked eye. In one case an intraocular tumor was studied by means of the slit lamp microscope in greater and sharper detail with the contact glass in place.

SLIT DIAPHRAGM CAP FOR SLIT OPHTHALMOSCOPY*

HARVEY E. THORPE, M.D.

PITTSBURGH

IN THE examination of the ocular fundus it is often advantageous to have focal illumination available. Wolff[1] focused the ophthalmoscope filament image on the retina, Koeppe[2] used a contact glass with the Gullstrand slit lamp and a monobjective binocular microscope, Friedenwald[3] devised a special ophthalmoscope.

To this end and for use with the May ophthalmoscope, I modified a pinhole diaphragm cap six years ago. This permits focal illumination of the retina. The diaphragm cap has a slit which can be focused sharply on the retina and thus affords focal and parafocal illumination of that structure.

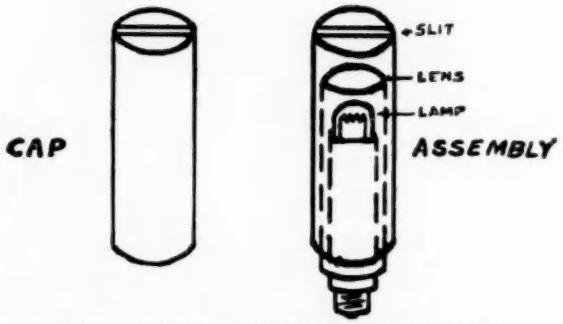

FIG. 1 — (Thorpe) — Slit diaphragm cap for the May ophthalmoscope.

The dimension of the slit which I found most useful in the past six years has been a width of .3 to .37 millimeters. I gave these dimensions to Bausch & Lomb Optical Company in April, 1930, and from time to time they have supplied ophthalmologists with such slit caps. There are definite advantages in studying the retina by parafocal or indirect illumination. When the sharp image of the

* This slit cap is manufactured by Bausch & Lomb Optical Co., Rochester, N. Y.

slit is focused on the retina it scatters light laterally in the retinal structures. Thus minute changes such as early senile changes in the pigment epithelium and the retina proper are seen more easily than in the ordinary ophthalmoscopic illumination.

Cystic degeneration and edema of the retina are recognized more easily with focal and parafocal (indirect or lateral) illumination. The method of using slit or focal illumination of the fundus does not do away with the necessity of the routine ophthalmoscopic examination.

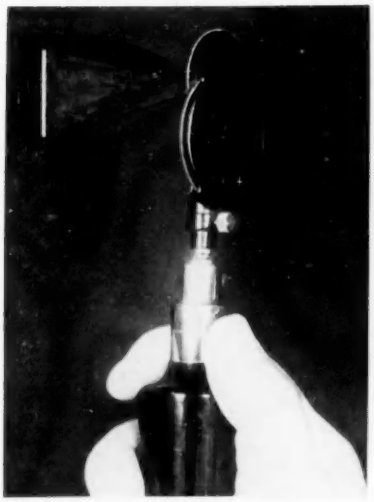

FIG. 2 — (Thorpe) — Slit beam as projected by May ophthalmoscope equipped with slit diaphragm cap.

The cap is 10 millimeters long, 5 millimeters inside diameter, and the slit measures 3 millimeters in length by .3 millimeters in width.

Similar caps with a slit .15 or .2 millimeters wide have been used by me with the illumination decentered. This is accomplished by setting the ophthalmoscope head so that the peephole is 3 to 5 millimeters off center with the illuminating axis.

In assembling the slit cap with the May ophthalmoscope, it is applied over the small lens cap and over the miniature lamp of the ophthalmoscope. The slit must, of course, be parallel with the long axis of the filament. It is adjusted so that the beam projected

from the ophthalmoscope is vertical. This avoids the color dispersion caused by the prism. The vertical slit beam is then focused at ½ meter distance.

With the ophthalmoscope thus equipped one can sweep across from the disk laterally to the macula and to other portions of the fundus and observe it by focal and parafocal illumination.

1. Wolff, H.: Zeitschr. f. Augenheilk., Bd. 29, S. 229, 1913.
2. Koeppe, L.: *Die Mikroskopie des Lebenden Auges*, Bd. 2.
3. Friedenwald, J.: Tr. Am. Oph. Soc., Vol. 26, p. 381, 1928.

OTOLARYNGOLOGY

PLASTIC REPAIR OF FRONTAL DEFORMITY USING HUMP FROM NOSE

C. R. STRAATSMA, M.D.

NEW YORK

THE COLORED FILM which was accompanied by sound demonstrated the repair of a depression over the left frontal sinus by utilizing the hump from the nose. This deformity resulted from an external operation for acute suppurative frontal sinusitis complicated by osteomyelitis of the outer table. Clinically, the case was cured by obliteration of the sinus. Skillful surgical intervention saved the patient's life, but in so doing a depression had necessarily resulted. This deformity often becomes increasingly prominent in the mind of the patient so that a mental illness frequently follows a physical one.

Plastic surgery was then called upon to repair the resulting defect and thus supplemented the work of the nose and throat surgeon.

A number of substances may be used to fill in depressed cavities; such as cartilage, bone, fat and fascia, or the dermal graft. Foreign material, such as ivory, celluloid, etc., is to be recommended only in selected cases because in spite of instances where such substances have remained in situ for years without trouble, there is a greater tendency for them to slough out.

Upon receiving this case it was first made certain that ample time had elapsed since the last operation to completely eliminate all traces of the original disease. An interim of several months is advisable. When considering the various sources of material for repair it occurred to us that the nasal hump might be removed and used to fill in the depression. This would reduce the size of the nose as well as lessen the apparent depth of the hollow and the

hump, being large, would also supply suitable and ample material with which to fill the defect.

The removal of the excess nasal bone was done through an intranasal incision along the lower border of the upper lateral cartilage and after separating the soft tissues from the nasal framework the excess was removed. After carefully elevating the soft parts from the inner table of the left frontal sinus, the hump, as removed from the nose, was inserted under the skin to fill the defect. The original scar was excised and the skin edges sutured with silk. The remaining bony framework of the nose was then narrowed and a pressure dressing applied over the nose and the left frontal sinus region, thus completing the entire repair.

In dealing with cases of similar nature it should be very comfortable for the rhinologist to feel that he could remove all the diseased structures necessary to effect a cure and then call upon reparative surgery to mend the resulting deformity.

COLOR MOTION PICTURES OF THE LARYNX

FRANCIS E. LEJEUNE, M.D.

NEW ORLEANS

VISUALIZATION of laryngeal lesions by means of motion pictures creates an indelible impression which is retained over a long period of time. Color motion pictures showing normal structures and pathological conditions existing within the laryngeal cavity are of inestimable value provided there is faithful color reproduction. As a teaching agent, color motion pictures can only be surpassed by a deliberate mirror examination of the clinical case. In teaching large groups, this type of individual examination by the students is difficult of accomplishment. In many cases it is highly desirable to have an accurate record in normal colors of the lesion in question.

The color motion pictures presented to the Academy represents clinical material collected over a period of time. While black and white motion pictures of the larynx present a certain amount of difficulty, the problems encountered in color photography were much greater. Obviously, if the color pictures are to have any lasting merit for teaching purposes, duplicate prints of the original must be available. Only the Morgana method permitted this necessary requisite in color photography. All views of the larynx were obtained by means of the Suspension Laryngoscope. The first few views presented were of a normal larynx. The normal anatomical structures were hurriedly reviewed and the usual favorite sites for the various types of lesions were pointed out. Numerous views showing benign lesions occurring within the larynx were presented. Motion pictures permit a deliberate pre-operative as well as a post-operative study of many lesions. In practically all cases an attempt was made to show the lesion before and immediately after surgical intervention. Many views of multiple papilloma of the larynx, ranging in size from a small growth to a mass completely filling entire larynx, were presented for observation and study. Multiple papilloma occasionally occurs in adults and several such lesions were shown on the screen.

One of the greatest factors of color motion pictures of the larynx is the forcible realization to those outside of our specialty that persistent hoarseness is the danger signal of the larynx. With this realization, an earlier diagnosis of malignant conditions occurring within the larynx must be expected. Many of these cases in their early stages are amenable to treatment. Numerous views of intrinsic carcinomas were presented and the results of intralaryngeal surgery discussed. Properly selected cases give brilliant results. Lesions of advanced malignancies in which surgery was contraindicated were studied.

Two views of foreign bodies in situ, one of a piece of beef-bone embedded in hypopharynx and the other of a chicken bone wedged in the larynx, were presented for observation. To the best of my knowledge these represent the first actual photographs of foreign bodies in such localities.

Motion pictures afford an excellent opportunity for the study of the physiology of the larynx and space only prevents the recording of interesting phenomena observed.

UNILATERAL AND BILATERAL ELIMINATION OF LABYRINTH IN GUINEA PIGS

HENRY DINTENFASS, M. D.
PHILADELPHIA

CAESAR HIRSCH, M.D.
NEW YORK

IF CERTAIN drugs, such as cocain, pontocain, chloroform, or nupercain, are introduced into the ear canal of a guinea pig, the corresponding labyrinth can be temporarily completely eliminated.

There is an anatomic sequence in the paralysis of the labyrinth. Three stages of the paralytic process may be distinguished depending on the anatomical structure of the labyrinth. The saccule is first affected because of its proximal position in the path of the drug. Paralysis of the left saccule results in the elimination of the labyrinthian righting reflex when the animal is placed on its left side. The second stage shows paralysis of the utricle producing basal rotation of the body. In the third stage, the drug reaches the semicircular canals, paralysis of which is followed by turning of the head and nystagmus of the eyes and head.

After five minutes the righting reflex is normal when the animal is placed on the right side. The righting reflex is absent when the animal is placed on its left side because of the paralysis of the left saccule. Due to paralysis of the utricle a rotation of the entire body around the longitudinal axis towards the left occurs. This is called basal rotation and is caused by the disturbance of the connection of the utricle with the homolateral muscles of the neck and body.

The otoliths and membrane of the utricle are shown displaced by centrifugation. Caloric tests show that the normal reaction of the semicircular canals is still present. Ethyl chloride is sprayed into the left ear of a normal animal to demonstrate the same reaction.

A few minutes after the paralysis of the utricle the semicircular canals are affected. This is manifested by turning of the head

towards the left side turning of the body around its vertical axis with nystagmoid movements of the eyes.

In an X-ray of the animal's body in situ we see that the curve involves the entire thoracic vertebral column with a slight scoliosis toward the right side and a torsion, i.e., rotation of the whole spine.

The labyrinthian righting reflex of the extremities can be corrected by placing the head of the animal in the straight and overcorrected straight position.

The peak of the reaction following the introduction of the chemical into the left ear is obtained when rolling movements of the animal begin. The animal throws itself to the side of the affected labyrinth. The rolling movements in the exhausted animal are less rapid. Fifteen minutes after the peak of the reaction the rolling movements stop. When both labyrinths are eliminated simultaneously the equilibrium is very much disturbed and the animal becomes perfectly limp. In bilateral elimination of the labyrinth with the right side eliminated two hours after the left side a less intensive reaction results with all the movements to the right side.

The normal guinea pig has a jumping reflex with the proper spreading of the toes and extension of the legs. This "readiness to jump" is absent in the animal with bilateral labyrinthian paralysis and the animal falls to the floor with a thud. The lift reflex is absent in an animal with bilateral labyrinthian paralysis.

A purulent labyrinthitis was produced in a cat by injecting croton oil into the left labyrinth. Physostigmin was injected to induce severe salivation. The cat, in cleaning its mouth, past-pointed to the left in accordance with the direction of the slow component of the nystagmus resulting from the elimination of the left labyrinth.

THE PRODUCTION OF THE PSEUDO-VOICE AFTER TOTAL LARYNGECTOMY

WALLACE MORRISON, M.D.
and SOLOMON FINEMAN, M.D.

NEW YORK

TWO METHODS obtain to provide speech in a laryngectomized patient: (1) A mechanical artificial larynx. (2) Pseudo-voice. Most, if not all, are familiar with the artificial larynx. Today we elect to show laryngectomized patients who have developed the ability to speak, and to discuss methods by which speech is developed.

A moment's thought regarding the laryngectomized patient will make it plain that he has lost not only the larynx, which is the essential organ of sound production for speech, but that his lungs can no longer act as bellows to supply air for phonation, since such air now passes in and out of the lungs through the tracheotomy tube or tracheal stoma. If a pseudo-voice is to be produced, it is obvious that Nature must supply not only some organ to take the place of the vocal cords — a pseudo-glottis where sound vibrations may be set up — but also a bellows and air reservoir to supply the stream of air necessary to produce speech; this must act as a vicarious lung into which air can be drawn and then expelled to set the pseudo-glottis in vibration.

The requirements for the proper functioning of the pseudo-glottis are that it must consist of some part which can be set into audible vibrations by a current of air passing out of the reservoir below; that is, it must be an area which can be voluntarily constricted. The sites at which the vicarious pseudo-glottis can form are between the base of the tongue and the posterior pharyngeal wall, between the back of the tongue and the stretched velum, between strongly contracted posterior palatine arches of both sides, between contracted portions of the inferior pharyngeal constrictor, between the epiglottis and the lateral or posterior pharyngeal wall, and at the upper hiatus of the esophagus between folds formed by the cricopharyngeus muscle.

The requirements for the vicarious lung or air reservoir are that it must be a cavity which will contain more than a few cubic centimeters of air, which can voluntarily be filled with air at a rapid rate, and from which the air can be voluntarily expelled; and that the air so expelled will be set into audible vibration as it passes the pseudo-glottis. The possible sites of such an air chamber are the mouth, the pharynx, especially the hypopharynx, the esophagus, particularly its cervical and upper thoracic portions, and the cardiac end of the stomach which normally contains the stomach bubble of air.

True pharyngeal pseudo-voice is one in which there is a real pseudo-glottis somewhere in the oropharynx, and an air reservoir in the depths of the hypopharynx, into which air is drawn by suction or swallowing, and expelled by voluntary contraction of the muscular walls. This type of pseudo-voice permits of vowel and consonant formation and the voice, while very gutteral and hoarse, is usually distinct and useful. The voice is quite distinct if the patient aspirates air quietly, and articulates distinctly.

True esophageal and stomach pseudo-voice is characterized by the fact that the pseudo-glottis lies at the upper end of the esophagus at the level of the cricopharyngeus muscle, and that the air reservoir is within the lumen of the esophagus, and the cardiac end of the stomach. Air in quite large quantities is taken into the reservoir by swallowing, brought out again by belching, and the resultant sound vibrations produced by the pseudo-glottis changed into vowels and consonants, syllables and words by the usual mechanism of articulation. This is the pseudo-voice in its most perfect form. When it has been learned, and practiced long enough, it is produced with little effort, is loud and clear enough for all ordinary conversation and carries very well by telephone, is rather low-pitched and monotonous, but no worse than the voice produced by a patient with an ordinary acute laryngitis. It serves the patient in carrying on his business, and in all social contacts, and frees him for life from the severe handicap of being voiceless.

A laryngectomized patient should be helped to develop a useful pseudo-voice as soon as the operative wound is soundly healed — usually within four to six weeks after operation. Actual instruction in forming the pseudo-voice begins with a discussion with the patient of the desirability of learning to produce a good voice, with

the reasons why he is no longer able to produce a sound, and of the reason why he must begin at once to remember that ordinary respiration no longer has anything to do with production of voice for him, but that he must understand that he will have to learn to make whatever sounds he can with the pseudo-voice, *between* breaths into and out of the lungs. Something of the theory of the production of the pseudo-voice must be explained to him, so that he will understand his problems and what is to be done for him. The author does this by means of verbal description, aided by a series of diagrams illustrating the points mentioned, and for study by the patient, typewritten material explaining fully and in non-technical language all that has been discussed.

The next step is to have the patient begin to practice the swallowing of air, always *between breaths*. He is told to swallow a very big mouthful of air, as deeply as possibly, without moving the chest wall. To help him, he pretends to drink from a pint-sized empty glass. The finger tip may be placed over the tracheal tube while he swallows to remind him that he must not breathe in or out while he swallows air. Practice is done in periods of not more than ten minutes at a time, six or more times per day, with the patient watching his movements in a large mirror.

The next step is to have the patient belch forth the swallowed air, producing the loudest "ructus" possible. He must learn to swallow and belch quickly, and to repeat the process over and over in steady succession. He will be helped to acquire the "feel" of this by drinking small quantities of plain charged water and belching up the gas from the stomach.

When the patient can swallow and belch more or less at will he is given syllables and short words, which he attempts to produce by ordinary articulation from the belching sound made with eructed air. Gutteral syllables are easiest, and most patients first learn to say "good," "god," or "gut." Practice is continued with the consonants "p, b, t, d, k, and g," to which are added the various vowel sounds. At first, each syllable so formed is made with one swallow and belch, until short words, first one or two, then four or five, are made with each swallow and belch. The patient now has really attained a pseudo-voice; he can carry on with constant practice, increasing his facility in swallowing and forming sounds into words and sentences, adding to his vocabulary by daily use of the voice, first with friends, then in all surroundings.

Not every laryngectomized patient can learn to produce a good pseudo-voice. To learn, there must be intelligence that permits understanding what is to be done, physical strength to carry on the exercises, and often much patience and perseverence in overcoming difficulties. Some will not try at all; others become discouraged after a few trials and cease trying; for these the pseudo-whisper or the use of the artificial larynx is the only means of making themselves understood by those about them. With understanding and a will to learn that does not yield at moderate discouragement, the pseudo-voice is within the reach of anyone who needs it.

LARYNGECTOMY . . . LARYNGEAL TUMORS

GORDON B. NEW, M.D.

ROCHESTER, MINN.

THE MOTION PICTURE on benign tumors illustrates the use of suspension laryngoscopy in removing these tumors, as was brought out by Lynch years ago. It also illustrates the use of surgical diathermy in removing the benign tumors and the early small malignant lesions.

The motion picture on larygectomy shows the technic of this operation, bringing out particularly the method of suturing the trachea and the tongue-shaped flap obtained from the inside of the cricoid to the skin; also the use of the artificial larynx in those cases that can not produce speech by means of the pharyngeal muscles.

THE TECHNIQUE OF BRONCHOSCOPY

JOEL J. PRESSMAN, M.D.

LOS ANGELES

THIS motion picture, produced in sound, presented the technique of bronchoscopy. It outlined the construction of essential instruments and their method of use. Certain problems of foreign body removal were presented together with a demonstration of the causes of failure resulting from improper selection of forceps and from improper usage of correct forceps. These were contrasted with proper methods.

Included in the film were actual bronchoscopic photographs of various portions of the tracheo-bronchial tree as they appear to the examiner through the bronchoscope.

The film was produced as an attempt to present a didactic lecture, presenting throughout the speaker delivering his address as it would be delivered in a class room, and interrupting the lecture to illustrate each point of the text by a motion picture sequence. The purpose of this was to suggest a method by means of which important lectures by outstanding authorities could be presented in an intimate manner to groups of students in institutions other than the one in which the teacher lectured. Thus, for instance, lectures by good men in absentia could be presented to students in out of the way schools where facilities for teaching bronchoscopy were inadequate. The personal touch and stimulation which results from hearing and seeing our outstanding personalities are preserved by their appearance on the screen, and facilities for vivid illustration unsurpassed by any other method.

Minutes

The American Academy
of Ophthalmology and Otolaryngology

* * *

Forty-First Annual Meeting
*New York * 1936*

MINUTES

AMERICAN ACADEMY *of* OPHTHALMOLOGY *and* OTOLARYNGOLOGY

* * *

MONDAY, SEPTEMBER 28, 1936, 9:20 A.M.

Joint Session

THE JOINT SESSION convened at nine-twenty o'clock, Monday morning, September 28, 1936, in the Grand Ballroom, Waldorf-Astoria Hotel, New York City, the President, Dr. Frank E. Burch, St. Paul, Minnesota, presiding.

THE PRESIDENT: I now declare this forty-first meeting of the Academy open. First, I wish to introduce Dr. Marvin F. Jones, the Chairman of the Committee on Arrangements, who will make the announcements for the convention. Dr. Jones! (Dr. Marvin F. Jones made several announcements.)

President Frank E. Burch read his Presidential Address, the First Vice-President, Dr. Gordon F. Harkness, Davenport, Iowa, presiding.

Sir John Herbert Parsons, London, England, the Guest of Honor, read his address on "Ophthalmology and Research."

A symposium on "Manifestations of Diseases Affecting Cerebral Nerves Supplying Eye, Ear, Nose and Throat due to Involvement of the Central Nervous System" was participated in by: Dr. D. J. Lyle, Cincinnati, Ohio; Dr. Marvin F. Jones, New York City, and Dr. H. A. Riley (by invitation), New York City.

The Joint Session adjourned at one-ten o'clock.

MONDAY, SEPTEMBER 28, 1936, 2:15 P.M.

Section on Otolaryngology

The first meeting of the Section on Otolaryngology convened at two-fifteen o'clock, Monday afternoon, September 28, Dr. Gordon F. Harkness presiding.

Dr. Lee W. Dean, of St. Louis, Missouri, read a paper (written in conjunction with Drs. H. M. Smit, Charles Mahoney, L. W. Dean, Jr., and Lloyd D. Linton) entitled, "The Treatment of Allergic Rhinitis with Particular Reference to Ionization and the Control of Chronic Vasomotor Rhinitis." Discussed by Drs. French K. Hansel, of St. Louis, Missouri, and Dr. Ralph A. Fenton, Portland, Oregon.

Dr. Lawrence R. Boies, Minneapolis, Minnesota, read a paper entitled, "Extradural Inflammation — A Study of Its Occurrence in Acute Surgical Mastoiditis." Discussed by Dr. H. P. Mosher, Boston, Massachusetts.

Dr. Robert H. Ivy, of Philadelphia, Pennsylvania, read a paper entitled "Tumors and Cysts of the Mouth and Jaws of Interest to the Otolaryngologist." Discussed by Drs. V. H. Kazanjian, Boston, Massachusetts; Fred Z. Havens, Rochester, Minnesota, and E. F. Risdon, Toronto, Canada.

Dr. Isidore Friesner, of New York City, read a paper entitled, "Report of Twenty-Four Cases of Petrositis." Discussed by Drs. Ernest M. Seydell, Wichita, Kansas, and Robert L. Moorhead, Brooklyn, New York.

Dr. Olof Larsell (by invitation), Portland, Oregon, read a research report on "Sympathetic Innervation and Lymphatic Pathways in the Nose." Dr. Ralph A. Fenton, Portland, Oregon, read a research report on "Substances Resembling Ephedrin."

The session adjourned at five-ten o'clock.

MONDAY, SEPTEMBER 28, 8:00 P.M.

Teachers' Section

The Dinner Meeting of the Teachers' Section, being a Panel Discussion on "Graduate Medical Education," convened in the Empire Room of the Waldorf-Astoria Hotel, New York City, at eight o'clock, Monday evening, September 28, Dr. L. W. Dean, St. Louis, Missouri, Chairman of the Section, presiding.

Dr. Willard Cole Rappleye, of New York City, presented the introductory remarks entitled "The Present Situation in the Medical Schools Regarding Graduate Instruction," during the presentation of which Dr. Dean entered into a short discussion with him on some of the points brought out.

Dr. Ralph A. Fenton, of Portland, Oregon, spoke on "Shall a Nine Months' Course in the Basic Sciences of Otolaryngology Be Required?" After the presentation there was a short discussion on the subject.

Dr. Dean M. Lierle, Iowa City, Iowa, discussed the subject of "The Amount of Training in the Basic Sciences of Otolaryngology that Each Interne Must Receive."

Dr. C. S. O'Brien, Iowa City, Iowa, presented the subject of "Shall a Nine Months' Course in the Basic Sciences of Ophthalmology Be Required?" During the presentation of his subject, Dr. Dean entered into a discussion with Dr. O'Brien concerning certain phases of it. The subject was also discussed briefly by Dr. G. M. Coates, of Philadelphia, Pennsylvania, who spoke regarding the courses given in Philadelphia.

Dr. Harry S. Gradle, Chicago, Illinois, next spoke on "The Amount of Training in the Basic Sciences of Ophthalmology that Each Interne Must Receive."

Sir John Herbert Parsons, Guest of Honor of the Academy, spoke briefly on the qualification of Ophthalmologists in England.

Dr. Harris P. Mosher, Boston, Massachusetts, spoke on "The Educational Standard of Otolaryngology that Must Be Met at this Time," commenting during his talk on some of the points brought out by other speakers.

Dr. Walter B. Lancaster, Boston, Massachusetts, then spoke on "The Educational Standard of Ophthalmology that Must Be Met at this Time."

The final presentation was made by Dr. W. D. Cutter, Secretary of the Council on Medical Education, American Medical Association, who commented on the presentations of the previous speakers, as well as expressing some of the views of the Council on the subject of "Graduate Medical Education."

After expressing the thanks of the Section to those participating in the discussion, Dr. Dean adjourned the session at 10:15 p.m.

TUESDAY, SEPTEMBER 29, 2:30 P.M.

Section on Ophthalmology

The first session of the Ophthalmological Section convened in the Grand Ballroom, Hotel Waldorf-Astoria, at two-thirty, Tuesday afternoon, September 29, President Burch presiding.

Dr. Alan Woods, Baltimore, Maryland, read a paper entitled, "Allergy in Clinical Ophthalmology." Discussed by Dr. A. C. Hilding, Duluth, Minnesota.

Dr. S. Hanford McKee, Montreal, Canada, read a paper entitled, "Sarcoma of the Uveal Tract — Malignant Melanoma." Discussed by Dr. T. L. Terry, Boston, Massachusetts.

Dr. A. J. Bedell, Albany, New York, read his paper entitled, "Pigmentation of the Fundus." Discussed by Drs. W. E. Fry, Philadelphia, Pennsylvania, and T. L. Terry, Boston, Massachusetts.

Dr. A. B. Reese, New York City, read a paper entitled "Defective Central Vision Following Successful Operations for Detachment of the Retina." Discussed by Drs. Clifford B. Walker, Los Angeles, California, and Harry S. Gradle, Chicago, Illinois.

The session adjourned at five-thirty o'clock.

TUESDAY, SEPTEMBER 29, 9:10 P.M.

Section on Instruction

The panel discussion on "Lymphatic Drainage of the Head" was held in the Sert Room of Hotel Waldorf-Astoria, on Tuesday evening, September 29th, at nine-ten o'clock, Dr. Harry S. Gradle, Chicago, Illinois, Chairman of the Section on Instruction, presiding.

Dr. Oscar V. Batson, Philadelphia, Pennsylvania, read a paper entitled, "Lymphatic Drainage of Head and Neck as Shown by Injections of Infant and Adult Human Material, with Clinical Comments."

Dr. A. G. Pohlman, Omaha, Nebraska, (by invitation) read a paper entitled, "Lymphatic Drainage of Labyrinth and Neighboring Areas, with Clinical Comments."

Dr. Olof Larsell, Portland, Oregon, (by invitation) read a paper entitled, "Experimental and Microscopic Evidence on Lymph Pathways from Accessory Sinuses."

The session adjourned at ten-ten o'clock.

WEDNESDAY, SEPTEMBER 30, 2:10 P.M.

Section on Otolaryngology

The second session of the Section on Otolaryngology convened in the Grand Ballroom of the Waldorf-Astoria Hotel at two-ten p.m., Wednesday, September 28th, Dr. Gordon F. Harkness, First Vice-President, presiding.

Dr. Hayes E. Martin, New York City, read a paper (by invitation) entitled, "Cancer of the Larynx." Discussed by Drs. Gabriel Tucker, Philadelphia, Pennsylvania; Joseph C. Beck, Chicago, Illinois, and John L. Myers, Kansas City, Missouri.

Dr. John J. Shea, Memphis, Tennessee, read a paper entitled, "Indications for Blood Transfusion in Otolaryngological Work." Discussed by Drs. R. E. Stetson, New York City; J. G. Dwyer, New York City; J. J. Dowling, Albany, New York, and A. A. Eggston, New York.

Dr. Joseph A. Sullivan, Toronto, Canada, read a paper entitled "Modification of Ballance-Duel Technic in the Treatment of Facial Paralysis." Discussed by Drs. John R. Page, New York City; Thomas G. Tickle, New York City, and E. P. Fowler, Jr., New York City.

Dr. Joel J. Pressman, Los Angeles, California, read a paper written in conjunction with Dr. Clyde K. Emery, Los Angeles, California, entitled, "A New Method of Radium Application in Cancer of the Bronchus." Discussed by Drs. Louis H. Clerf, Philadelphia, Pennsylvania, and Francis E. LeJeune, New Orleans, Louisiana.

The section then adjourned at five o'clock.

THURSDAY, OCTOBER 1, 2:10 P.M.

Section on Ophthalmology

The second session of the Section on Ophthalmology convened in the Grand Ballroom of Hotel Waldorf-Astoria, New York City, on Thursday afternoon, October 1st, at two-ten p.m., President Burch presiding.

Dr. Otto Barkan, San Francisco, California, read a paper entitled, "An Operation for Chronic Glaucoma: Restoration of Physiological Function by Opening Schlemm's Canal Under Direct Magnified Vision." Discussed by Drs. John M. Wheeler, New York City; M. Uribe Troncoso, New York City; Harry S. Gradle, Chicago, Illinois, and Sir John Herbert Parsons, London, England.

Dr. Parker Heath, Detroit, Michigan, read a paper entitled, "Visual Sequelæ from Meningococcus Meningitis." Discussed by Dr. Phil M. Lewis, New Orleans, Louisiana.

Dr. Benjamin Rones, Washington, D. C., read a paper entitled, "Nodular Dystrophies of the Cornea." Discussed by Dr. Mark J. Schoenberg, New York City.

Drs. Alfred Cowan and Wilfred E. Fry, Philadelphia, Pennsylvania, presented a paper (Dr. Fry reading) entitled, "Elschnig Bodies, with Particular Reference to their Occurrence in Secondary Cataract." Discussed by Dr. Harry S. Gradle, Chicago, Illinois.

Dr. Bennett Y. Alvis, St. Louis, Missouri, read a paper (prepared in collaboration with Dr. Meyer Wiener, St. Louis, Missouri) entitled, "The Use of Concentrated Epinephrine Preparations in Glaucoma, Iritis and Related Conditions — a Clinical Study." Discussed by Dr. Ed. C. Ellett, Memphis, Tennessee.

Dr. K. L. Stoll, Cincinnati, Ohio, read a paper entitled, "Glaucoma and Nevus Flammeus." Discussed by Drs. Arnold Knapp, New York City; Ernest F. Krug, New York City; E. B. Dunphy, Boston, Massachusetts, and F. Bruce Fralick, Ann Arbor, Michigan.

Dr. Frederick A. Kiehle, Portland, Oregon, read a paper entitled, "Report of a Case of Scleromalacia." Discussed by Dr. William L. Benedict, Rochester, Minnesota.

The session adjourned at five-thirty o'clock.

THURSDAY, OCTOBER 1, 5:30 P.M.

Business Meeting

The annual business meeting of the American Academy of Ophthalmology and Otolaryngology convened at five-thirty o'clock, Thursday afternoon, the President, Dr. Frank E. Burch, presiding.

The following reports were read:

REPORT OF EXECUTIVE SECRETARY-TREASURER

The membership summary as of September 1, 1936, was as follows:

Honorary Members	21	
Life Members	130	
Senior Members	98	
Junior Members	1930	2,179
Candidates		221
Total carried on books		2,400

During the year 31 deaths were reported which will be summarized by the Historian.

Those delinquent in payment of dues, September 1st:

1936	78
1935	32
1934	35
1933	31
1932	25
1931	20
	221

Many of the delinquents listed in this number (221) have since September first lifted the delinquency. It may be of interest to note the delinquent list at the time of the 1935 convention was 264 as contrasted to 221 for the current year.

The following resignations have been accepted by the Council:

George Ainslie, Portland, Oregon
J. A. Connell, Riverside, California
F. G. Fielding, Glens Falls, New York
J. H. Laning, Kansas City, Missouri
Dennis J. McDonald, New York City, New York
Lillian B. Mueller, Indianapolis, Indiana
A. N. Murray, Chicago, Illinois
William S. Newell, Washington, D. C.
G. A. Sulzer, Columbus, Ohio
C. F. Welty, San Francisco, California
Orton E. White, Cortland, New York

A report of the Auditor for the year 1935 is appended as a part of this report.

Summarized briefly: The gross income was $37,220.14; allocated in part to dues and applications, $20,332; Transactions, $898.49; Convention, $11,689.75. The disbursements were $32,939.06; allocated in part to research, $2,604.11; Transactions, $5,965.85; Convention, $15,391.80; General office operation, $6,877.30; Honorariums, $2,000. A surplus of $4,381.08 was added to the Investment Account. The cost of the Transactions was $2.53 per volume.

During the current year — January 1st to September 1st — very careful attention has been given the portfolio of investments making up the Academy Capital Account. This has been made necessary since many corporations are refinancing their obligations. During this period — eight months — over $30,000 in sales and over $31,000 in reinvestments have been handled. The differential between the cost and sales value netted the Academy over $1,600 in profit, but with an interest loss of about $300. Obviously this favorable balance cannot be expected in all contrast periods.

An analysis of the portfolio, excepting from the contrast three items that some day probably will be wiped out and that have been carried for some fourteen years, shows an increase of approximately $800 market value over cost value as of September 1, 1936.

Financially, the Academy has little to complain about incident to the depression.

A rather grave problem is before the Academy, namely, where to meet and preserve the intimacy so characteristic of Academy conventions. The expansion of activities has made it such that few hotels in the country can accommodate us, yet to change the plan and transfer the meeting places to civic auditoriums, seemingly would make of the convention just another big, unsociable, medical meeting, a program rather undesirable to the present secretarial management group. A place to meet, therefore, and to properly conduct the present Academy requirements offsets quite forcibly the desire to accept invitations to certain cities otherwise qualified.

The ever-increasing influence of the Academy and the growing expectancy that the Academy should take an active part in national medical affairs of interest as to the specialties has accentuated the necessity that members keep the executive office informed of matters in their communities. Cooperation of this sort will permit of cohesive action when necessary.

For the Secretarial Group, I desire to express an appreciation to the retiring officers for the help and advice during the current year.

Respectfully submitted,

W. P. Wherry, M.D.
Executive Secretary-Treasurer

REPORT OF THE AUDITOR

Omaha, Nebr., January 2, 1936

THE AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Gentlemen:

I have examined the accounts of your Academy for the period from January 1, 1935, to December 31, 1935. I submit herewith a summary of receipts and disbursements, together with a list of securities owned December 31, 1935.

The ordinary general expenses for the month of December have not been paid.

Office furniture and instruments have not been capitalized.

The bonds owned by the Academy are in possession of the Trust Department of the Omaha National Bank for safekeeping. They were verified by a certificate from the Bank. The securities are carried at cost, with no adjustments for amortization of premium or discount. Interest coupons maturing during the year amounted to $5,542.11, of which $3,962.11 was received, leaving $1,580.00 in default.

The changes in the financial status of the Academy during the year are accounted for as follows:

Assets held January 1, 1935		$110,527.27
Add: Excess of Receipts over Disbursements:		
Receipts	$37,220.14	
Disbursements	32,839.06	4,381.08
		$114,908.35

I certify that the Statements are correct to the best of my knowledge and belief.

Respectfully,

JOHN P. BEGLEY,
Certified Public Accountant

RECEIPTS AND DISBURSEMENTS

JANUARY 1, 1935 TO DECEMBER 31, 1935

Cash Balance January 1, 1935		$ 4,745.55
Receipts:		
Applications, dues, etc.	$20,332.00	
Transactions	898.49	
Conventions	11,689.75	
Bond interest	3,926.50	

Dividends	5.00	
Exchange on checks	1.15	
Profit on bonds called or sold	367.25	
	$37,220.14	
On Capital Account:		
Bonds called or sold	3,910.00	41,130.14
		$45,875.69
Disbursements:		
Research	$ 2,604.11	
Transactions	5,965.85	
Honorariums	2,000.00	
Meetings and conventions	15,391.80	
Office and general expense	6,877.30	
	$32,839.06	
On Capital Account:		
Bonds purchased	5,259.44	38,098.50
		$ 7,777.19

SUMMARY OF ASSETS, DECEMBER 31, 1935

Cash Balance	$ 7,777.19
Securities Owned	107,131.16
	$114,908.35

BANK RECONCILEMENT

OMAHA NATIONAL BANK, OMAHA, NEBRASKA

Balance per Bank Statement, December 31, 1935 ..... $ 7,777.19

Last check issued was No. 461. No checks were outstanding.

Upon motion by Dr. Goar, seconded, the report was accepted and placed on file.

SCHEDULE OF SECURITIES OWNED DECEMBER 31, 1935

	RATE %	DUE	PAR VALUE	COST
American International	5½	1949	$ 2,000	$ 1,962.50
Brooklyn Edison Co. Gen. Mtg. Gold Bonds Series "E"	5	1952	3,000	2,910.00
Central Illinois Public Service First Mtg. Gold Bonds	5	1968	3,000	2,925.00
Chicago, Milwaukee, St. Paul & Pac. First Mtg. Gold Series "A"	5	1975	2,000	1,821.50
Chicago, Rock I. and Pacific Ry. First and Refund. Gold Bonds	4	1934	3,000	2,668.50
Citizens Building Co. of Cleveland First Mtg. S. F. G. Bonds and 50 shares of stock	6	1946	5,000	5,000.00
Columbia Gas & Electric Company Debenture Bonds	5	1952	5,000	4,997.50
Commonwealth Edison Company First Mtg. & Col. Tr. Ser. "C"	4½	1956	5,000	4,825.00
Denver Gas & Electric Company General Mortgage	5	1949	4,000	3,951.00
Detroit City Gas Company First Mtg. Gold Bonds Ser. "B"	5	1950	5,000	5,025.00
Dominion of Canada War Loan Twenty-Year Gold Bonds	5	1937	3,000	2,790.00
Goodyear Tire & Rubber Company First Mtg. and C. Tr.	5	1957	2,000	1,839.00
Inland Steel Company Series "B" Gold Bonds	4½	1981	5,000	4,825.00
Interlake Iron Corporation Gold Bonds Series "B"	5	1951	5,000	4,850.00
Kansas City, Fort Scott & Memphis Railway	4	1936	5,000	4,352.50
Kingdom of Denmark Gold Bonds	5½	1955	3,000	3,030.00
Kingdom of Norway	5	1963	2,000	1,995.00
Massachusetts Gas Co.	5	1955	2,000	1,970.00
Midvale Steel & Ordnance Co. Conv. Sinking Fund	5	1936	5,000	4,902.64
Missouri Pacific Railroad First and Refunding Mortgage	5	1978	2,000	1,934.00
Missouri Pacific Railroad First Mtg. Ref. Series 1	5	1981	3,000	2,850.00
Ophthalmic Publishing Company 5 shares of Stock $20 par			100	Gift
Public Service Co. Northern Ill. First and Refund.	4½	1981	5,000	4,875.00
Southern California Gas Company First Mtg. & Ref. Gold	4½	1961	5,000	4,775.00
Southern Pacific Company	4½	1981	5,000	4,837.50
U. S. Treasury Notes	3¼	1937	5,000	5,259.44
Van Sweringen Company First Mtg. & Col. Trust Sink. F.	6	1935	6,000	6,031.33
Witherbee Sherman Company First Mtg. Sink. Fund Series "A"	6	1944	5,000	4,925.00
Youngstown Sheet & Tube Company First Mtg. Series "A"	5	1978	5,000	5,003.75
			$110,100	$107,131.16

In Memoriam

Herbert Lee Alkire, Topeka, Kansas
Adolph Barkan, San Francisco, California
Franklin Pierce Capron, Providence, Rhode Island
William Cohen, Brooklyn, New York
Samuel Gordon Dabney, Louisville, Kentucky
James Edward Davis, McAlester, Oklahoma
Herbert Hershey Farnsler, Harrisburg, Pennsylvania
Edward B. Heckel, Pittsburgh, Pennsylvania
Thomas Beaver Holloway, Merion, Pennsylvania
C. Norman Howard, Warsaw, Indiana
Joseph Clarence Keeler, Philadelphia, Pennsylvania
Hugo Albert Kiefer, Los Angeles, California
Clarence King, Cincinnati, Ohio
James Joseph King, New York City, New York
William Otis McBride, Fort Wayne, Indiana
Thomas E. McConnell, New Kensington, Pennsylvania
George Joseph McKee, Pittsburgh, Pennsylvania
Charles L. Minor, Springfield, Ohio
David Aloysius Prendergast, Cleveland, Ohio
Louis Alfred Roller, Grand Rapids, Michigan
Calvin C. Rush, Johnstown, Pennsylvania
Edwin G. Rust, Cleveland, Ohio
Daniel Kerfoot Shute, Washington, D. C.
Herman B. Slotkin, Philadelphia, Pennsylvania
Stanley Sinclair Smith, Pittsburgh, Pennsylvania
Joseph Anthony Stockler, Reading, Pennsylvania
John Joseph Thomson, Mount Vernon, New York
William S. Tomlin, Indianapolis, Indiana
Roy Seymour Watson, Saginaw, Michigan
William Hamlin Wilder, Chicago, Illinois
Harry Ralph Wormley, Rockford, Illinois

REPORT OF THE HISTORIAN

Dr. James A. Babbitt read his report, after which the members arose in silent tribute.

Dr. Herbert Lee Alkire

Dr. Alkire was born on November 17, 1862, in Springfield, Illinois, and died on April 27, 1936, at Topeka, Kansas.

Receiving his preliminary education at the University of Kansas, he took his medical degree at Jefferson Medical College in Philadelphia, in 1887. After postgraduate work in the clinics of Philadelphia, New York, Boston, and Chicago, Dr. Alkire took up the practice of medicine in Logan, Kansas, in 1887, and later began the practice of his specialties in Topeka.

In 1904, Dr. Alkire was appointed Dean of Washburn Medical College, and was also Professor of Anatomy and Otology in that institution. He was a member of the staff of St. Francis Hospital.

Dr. Alkire was a member of the Kansas City, Missouri, Eye, Ear, Nose and Throat Society, a charter member of the Shawnee Medical Society, and a member of the American Academy of Ophthalmology and Otolaryngology.

He was a member of the First Congregational Church in Springfield, Illinois. Dr. Alkire was greatly interested in higher Masonic activities as a Shriner and member of the Topeka Scottish Rite bodies.

He is survived by his widow and a brother's family.

Dr. Adolph Barkan

Dr. Barkan, who died on August 28, 1935, at Baden-Baden, Germany, was born in Zurich, Switzerland, ninety years ago.

Graduating from the Medizinische Fakultät der Universität Wien, Austria, in 1866, with the degree of M.D. Dr. Barkan took up the practice of medicine in San Francisco in 1869.

Dr. Barkan was professor of the structure and diseases of the eye, ear and larynx at Stanford University School of Medicine, San Francisco, for many years and was Emeritus Professor at the time of his death. He was also former Professor of Ophthalmology and Otology at the Medical College of the Pacific and Cooper Medical College in San Francisco, and for many years eye and ear surgeon to the Lane Hospital in the same city.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the California Medical Association and the Pacific Coast Oto-Ophthalmological Society.

Dr. Barkan had also received the degree of LL.D., from Glasgow University and was very much interested in music.

His widow, three sons and a daughter survive him.

Dr. Franklin Pierce Capron

Dr. Capron was born in Cumberland, Rhode Island, on November 2, 1852, and died in Providence on December 16, 1935.

He received his preliminary education at the Nowry and Goff School in Providence, his A.B. degree from Brown University in 1877, and medical degree from the College of Physicians and Surgeons of Columbia University, New York, in 1879. Following a year of general practice in Yonkers, N. Y., Dr. Capron returned to Providence and took up the practice of his specialty.

Dr. Capron established the ear clinic at the Rhode Island Hospital, and was the first physician to lecture to nurses in that hospital. He was surgeon to the Eye, Ear, Nose and Throat Department of the Rhode Island Hospital from 1881 to 1897, and Consulting Physician to the same hospital since 1897.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Otological Society and the New England Ophthalmological Society, as well as state and medical societies.

Dr. Capron was a great traveller and had a large circle of medical friends both here and abroad.

He is survived by one daughter.

Dr. William Cohen

Dr. Cohen was born in New York City on March 10, 1890, and died in Brooklyn, New York, on September 10, 1935.

After receiving his medical degree from the Long Island College Hospital, Dr. Cohen took postgraduate work in his specialty at the Manhattan Eye and Ear Postgraduate Hospital, and began the practice of medicine in New York City in 1917.

At the time of his death, Dr. Cohen was assistant visiting ophthalmologist at the Gouverneur Hospital, and an assistant in the ophthalmological department of the dispensary of the Long Island College Hospital.

Dr. Cohen was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

He is survived by his wife and two sons.

Dr. Samuel Gordon Dabney

Dr. Dabney was born in Charlottesville, Virginia, on August 6, 1860, and died in Louisville, Kentucky, on October 15, 1935.

After graduating from the University of Virginia and receiving his medical degree from the Hospital College of Medicine in Louisville, Dr. Dabney took postgraduate work in New York, Vienna and Zurich, returning to take up the practice of medicine in Louisville, Kentucky, in 1882.

Dr. Dabney was for forty-five years connected with the University of Louisville School of Medicine and at the time of his death was Emeritus Professor of Otology, Rhinology and Laryngology. He was also on the staffs of the Louisville City Hospital and the Norton Memorial Infirmary.

He was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

His wife, son and daughter survive him.

Dr. James Edward Davis

Dr. Davis was born in Greenback, Tennessee, on May 17, 1879, and was killed in an automobile accident near Shawnee, Oklahoma, on December 20, 1935, in which accident his wife was also killed instantly and a daughter critically injured.

After graduating from the Hospital College of Medicine in Louisville, Kentucky, Dr. Davis began the practice of medicine in 1904 in Sweetwater, Tennessee. Later he took postgraduate work in New York and Nashville, Tennessee, and entered the practice of his specialties in McAlester, Oklahoma, where he became widely known both as a specialist and as a leader in civic and community life.

Dr. Davis was a member of the American Academy of Ophthalmology and Otolaryngology, Oklahoma Medical Association and Pittsburgh County Medical Association. During the World War he was a member of the Medical Advisory Board, District

No. 35 of Oklahoma, and a Captain in the Army, also a member of the Volunteer Medical Service Commission.

He was keenly interested in church work and an enthusiast in the Boy Scout movement.

Three daughters and one son survive him.

Dr. Herbert Hershey Farnsler

Dr. Farnsler was born on June 10, 1878, at Union Deposit, Dauphin County, Pennsylvania, and died on March 14, 1936, in Harrisburg, Pennsylvania.

Receiving his early education at Ursinus Academy and Ursinus College, he graduated in Medicine from the Medico-Chirurgical College of Philadelphia in 1904, and began the practice of medicine in Myerstown, Pennsylvania.

At the time of his death Dr. Farnsler was consulting surgeon to the department of ophthalmology and otolaryngology of Harrisburg Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and various local and state medical societies.

Dr. Farnsler was a thirty-second degree Mason and a Shriner of Zembo Temple.

He is survived by his wife, a daughter and a son.

Dr. Edward B. Heckel

Dr. Heckel, who died in Pittsburgh, Pennsylvania, on December 23, 1935, of pneumonia, was born there on January 30, 1865.

He was a graduate of Allegheny College, Meadville, Pennsylvania, and Bellevue Hospital Medical College, New York City, later doing postgraduate work at the New York Polyclinic.

Dr. Heckel was greatly interested in medical organization and in 1898 was President of the Pittsburgh Academy of Medicine, in 1905 President of the Allegheny County Medical Society, served two terms as President of the Medical Society of the State of Pennsylvania and was the continuous President of the Pittsburgh Ophthalmological Society for twenty-two years.

On several occasions Dr. Heckel was a member of the House of Delegates of the American Medical Association, in 1925 was

elected a member of the Board of Trustees and became its Chairman. In 1928 he was elected to the Chairmanship of the Section on Ophthalmology.

Dr. Heckel was a member of the American Academy of Ophthalmology and Otolaryngology, various special local and state societies, and a Fellow of the American College of Surgeons.

At the time of his death Dr. Heckel was ophthalmologist to the Allegheny General Hospital, the Pennsylvania Railroad, the Carnegie Steel Company, the United States Pension Board, and the Western Pennsylvania Hospital. He served as a member of the Board of Managers of the Allegheny County Industrial and Training School for Boys and was a member of the Board of Regents of the University of Pittsburgh. He was also a member of various Masonic societies.

Dr. Heckel was an outstanding man among his fellow specialists. Of a most genial and gracious manner, he will live long in the memory of his friends.

His widow and two daughters survive him.

Dr. Thomas Beaver Holloway

Dr. Holloway was born in Danville, Pennsylvania, on March 24, 1872, and died at his home in Merion, Pennsylvania, on August 18, 1936.

Dr. Holloway graduated from Lafayette College where he subsequently received the degrees of M.Sc. and D.Sc., and his medical degree from the University of Pennsylvania in 1897.

At the time of his death Dr. Holloway was Professor of Ophthalmology at the University of Pennsylvania, this position culminating a long record of distinguished hospital service. In 1914 he was Professor of Ophthalmology at the Philadelphia Polyclinic, in 1918 Professor of Ophthalmology in the University of Pennsylvania Graduate School of Medicine, from 1919 to 1924 Surgeon to the Wills Eye Hospital, in 1920 Vice-Dean for Ophthalmology at the University of Pennsylvania Graduate School of Medicine, and in 1922 Professor of Neuro-Ophthalmology at the Philadelphia General Hospital. He had also been Ophthalmologist for the Pennsylvania Institution for the Instruction of the Blind at Overbrook.

Dr. Holloway was a past president and for several years was Secretary-Treasurer of the American Ophthalmological Society and at one time edited the society's transactions; past president of

the Section on Ophthalmology of the Pan-American Medical Association; a member of the American Academy of Ophthalmology and Otolaryngology, the Société Française d'Ophthalmologie, and the Ophthalmological Society of the United Kingdom, and various other local and state societies. In 1918 he was a member of the House of Delegates of the American Medical Association and Chairman of the Section on Ophthalmology from 1929 to 1930. In 1932 he was elected a member of the Board of Directors of the National Society for the Prevention of Blindness.

Dr. Holloway was an indefatigable worker. Your Historian's impression of him was that of unceasing effort in hospital and literary service. Included among his notable contributions was that in association with Dr. George E. de Schweinitz "Pulsating Exophthalmos" in 1908.

His social activity included membership in the Philadelphia Country Club, the Penn Athletic Club and the Metabetchouan Fish and Game Club.

He is survived by his widow and one son.

Dr. Charles Norman Howard

Dr. Howard was born in Bethlehem, Pennsylvania, on September 23, 1875, and died in Warsaw, Indiana, on November 11, 1935.

He received his medical degree from George Washington University, Washington, D. C., in 1898, and did postgraduate work in New York City and Chicago, Illinois. After interning at the City Hospital, Blackwell's Island, New York, Dr. Howard took up the practice of medicine in Washington, D. C. Later he practiced his specialty in Warsaw, Indiana.

Dr. Howard was a member of the American Academy of Ophthalmology and Otolaryngology, past President of the Kosciusko County Medical Society and the Indiana Academy of Ophthalmology and Otolaryngology, and member of various local medical societies.

Dr. Howard's service to his country included participation in the Spanish-American War, where he was secretary to General Ludlow, and had charge of the money expended by the expeditionary forces in Cuba. He was also a Captain of the Medical Corps during the World War. He served on the staff of the Walter Reed Hospital in Washington from 1917-1919, during which time he made special studies of the sinuses in the influenza epidemic.

He was a prolific contributor to the various journals on subjects pertaining to his specialties, and was greatly interested in work for boys.

His wife and two sons survive him.

Dr. Joseph Clarence Keeler

Dr. Keeler, who died on September 17, 1935, at the Jefferson Hospital, Philadelphia, was born on February 10, 1871, near Doylestown, Pennsylvania.

He graduated from Jefferson Hospital and Medical College in 1896, and began the practice of medicine in Philadelphia. His postgraduate work included studies in Vienna in 1921, and with Robert Barany at the time of his visit to America.

Dr. Keeler became Professor of Otology at Jefferson Medical College in 1930, was attending otolaryngologist at the Germantown Hospital, and consultant at the Pottstown Hospital and Newcomb Hospital of Vineland, New Jersey.

He was a member of the American Academy of Ophthalmology, the American Otological Society, the American Laryngological, Rhinological and Otological Society, the Philadelphia County Medical Society, and a Fellow of the American College of Surgeons, and the College of Physicians of Philadelphia.

During the World War, Dr. Keeler served in Southern training camps with the rank of Captain in the Medical Corps.

He was the author of several scientific books and a prolific contributor to medical literature. He impressed his associates by the deeply conscientious character of his hospital service. His hobbies were horses, dogs and hunting.

His widow, two brothers and two sisters survive him.

Dr. Hugo Albert Kiefer

Dr. Kiefer, who died in Los Angeles, California, on October 26, 1935, was sixty-five years of age.

He graduated from the University of Pennsylvania Medical School in 1897, and practiced his specialty in California.

Dr. Kiefer was formerly Adjunct Professor and Assistant Professor in Ophthalmology at the University of California, Los Angeles, and was on the visiting staff of the Hollywood Clara Barton Memorial Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology and the Pacific Coast Oto-Ophthalmological Society, and a Fellow of the American College of Surgeons.

Dr. Clarence King

Dr. King, who was drowned in Bermuda on August 15, 1936, at the age of fifty-seven, was a native of Newport, Kentucky.

He graduated from the Medical College of Ohio, Cincinnati, in 1901, and followed this with special work in ophthalmology for two years in the Universities of Vienna and Berlin.

At the time of his death Dr. King was Director of the Ophthalmic Service at the Children's Hospital, attending surgeon to the Cincinnati General Hospital and Director of the Outdoor Clinic, and Ophthalmologist to the Christ Hospital. At various times he had served on the staffs of the Ophthalmic Hospital, Good Samaritan Hospital and Deaconess Hospital, and was active in the establishment of the laboratory of ophthalmic pathology at the Christian R. Holmes Hospital.

Dr. King was a member of the Academy of Medicine of Cincinnati, a Fellow of the American College of Surgeons, a member of the American Academy of Ophthalmology and Otolaryngology and the American Ophthalmological Society, in which he held several important committee offices.

During the World War, Dr. King was a Major in the Medical Corps, rendering ophthalmological service at Allery Base Hospital, the Evacuation Hospital at Coincy, and Evacuation Hospital No. 4, at Coblenz, Germany, Army of Occupation.

He is survived by his widow, one son and one daughter, and a sister.

Dr. James Joseph King

Dr. King was born in Columbia, Tennessee, March 9, 1882, and died in New York City, November 29, 1935.

He graduated from the University of Tennessee in 1904 and received his medical degree from the University of Louisville in 1907. After interneship at the Children's Hospital, Philadelphia, and three years of intensive general practice, he was house surgeon at St. Bartholomew's Clinic, 1911-12, later became attending laryngologist to the Hospital for the Ruptured and Crippled and consultant in the Ear, Nose and Throat to the French Hospital.

He was a member of the New York Academy of Medicine, President of the Tennessee Society in New York for three terms, a member of the New York Southern Society and member of the American Academy of Ophthalmology and Otolaryngology. He was the author of "Local Anesthesia in Otolaryngology" and contributed to the current journals. He was Captain in the Medical Corps, attached to the air service during the War.

Dr. William Otis McBride

Dr. McBride was born in Joliet, Illinois, on December 24, 1878, and died in Fort Wayne, Indiana, on November 5, 1935.

He was a graduate of the University of Michigan and Northwestern Medical School, receiving his medical degree from the latter in 1907. He began the practice of medicine in Joliet, Illinois, later took a postgraduate course at the University of Vienna, and moved to Fort Wayne, Indiana.

He was a member of the American Academy of Ophthalmology and Otolaryngology, at one time president of the Fort Wayne Medical Society, at another Vice-President of the Indiana Academy of Ophthalmology and Otolaryngology, and was a member of other local and state medical societies.

Dr. McBride was oculist and aurist to the Duemling Clinic, and served the Lutheran Hospital in various capacities.

He is survived by his widow and two daughters.

Dr. Thomas Ethelbert McConnell

Dr. McConnell of New Kensington, Pennsylvania, died there in January, 1936, at the age of sixty-six.

He received his medical degree from Western Pennsylvania Medical College of Pittsburgh in 1891.

Dr. McConnell was a member of the Medical Society of the State of Pennsylvania and of the American Academy of Ophthalmology and Otolaryngology.

Dr. George Joseph McKee

Dr. McKee was born on February 9, 1880, in Cambridge, Massachusetts, and died on August 14, 1935, at Point Chautauqua, New York.

Receiving his preliminary education at Pomona College Preparatory School, Claremont, California, and Frey's Preparatory

School Boston, he graduated from Harvard Medical School in 1906, and served as Resident Physician at the Presbyterian Hospital, Pittsburgh, Pennsylvania, for two years, after which he began the practice of medicine in Pittsburgh. Desiring to specialize in ear, nose and throat work, Dr. McKee took postgraduate work with Dr. D. Braden Kyle at the Jefferson Hospital, Philadelphia, and later studied with Dr. Chevalier Jackson.

At the time of his death Dr. McKee was senior otologist to the Allegheny General Hospital and Columbia Hospital, both in Pittsburgh, and for about three years had been on the general staff of the Tuberculosis Dispensary at Pittsburgh, Pennsylvania.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of state and local medical societies.

Dr. McKee served on the Military Advisory Board during the World War.

He is survived by his widow, one daughter and one son.

Dr. Charles L. Minor

Dr. Minor was born in Mason, Ohio, on November 25, 1875, and died in Springfield, Ohio, on February 18, 1936.

He attended the Ohio State University and graduated in medicine from the Miami Medical College (University of Cincinnati) in 1897. For five years he was associated with Dr. Christian Holmes' Eye, Ear, Nose and Throat Hospital in Cincinnati, following which he took postgraduate work in Europe, and later moved to Springfield, Ohio.

Dr. Minor was a member of the American Academy of Ophthalmology and Otolaryngology, the Ohio State Medical Society, the Clark County Medical Society, and a Fellow of the American College of Surgeons.

He was on the staff of the Springfield City Hospital, chief of the medical staff of the Ohio Masonic Home, and a member of the Ohio Commission for the Blind.

He is survived by his widow and one daughter.

Dr. David Aloysius Prendergast

Dr. Prendergast, who died in Cleveland, Ohio, on December 2, 1935, at the age of fifty-four, received his medical degree from Western Reserve University Medical Department, Cleveland, in 1906.

At the time of his death Dr. Prendergast was visiting Otolaryngologist to St. John's Hospital, consulting otolaryngologist to the Lakewood Hospital, and Consulting Oculist to St. Ann's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons.

Dr. Louis Alfred Roller

Dr. Roller was born on February 23, 1855, in New York City, and died May 4, 1936, in Grand Rapids, Michigan.

After graduating from Rush Medical College, Chicago, with his medical degree, Dr. Roller took postgraduate courses in New York City and at the Chicago Polyclinic Hospital, and then began the practice of his specialties in Edmore, Michigan, in 1881.

Dr. Roller was a member of the American Academy of Ophthalmology and Otolaryngology, the Michigan State Medical Society, and for many years was a member and formerly president of the City Board of Health.

For a considerable period Dr. Roller was a member of the staff of the Butterworth Hospital in Grand Rapids, of which he was an honorary member at the time of his death.

He is survived by his widow, one daughter, four grandchildren and three great-grandchildren.

Dr. Calvin C. Rush

Dr. Rush was born in Fairmount, Indiana, on February 16, 1876, and died in Johnstown, Pennsylvania, on July 19, 1935.

After preliminary education in Earlham College and Haverford College, he received his medical degree from the University of Pennsylvania, Philadelphia. Dr. Rush did postgraduate work in the Wills Eye Hospital and Graduate Hospital of the University of Pennsylvania, Philadelphia, the Massachusetts Eye and Ear Infirmary, and then took up the practice of his specialties in Johnstown, Pennsylvania, in 1920.

He was at one time Assistant Demonstrator of Neuroanatomy at the Jefferson Medical College in Philadelphia; served on the staffs of hospitals in Canton, China, and Tokyo, Japan, and was affiliated with St. Luke's Hospital, in the eye and throat department, in 1918 and 1919. He was on the staffs of the Conemaugh

Valley Memorial Hospital and Mercy Hospital of Johnstown, Pennsylvania.

Dr. Rush was a member of the Society of Friends, was very much interested in work for the blind, in music, and all community affairs. Even with this brief review one is overwhelmed with the scope of service which Dr. Rush rendered in routine hospital work, the teaching department, and purely philanthropic affairs. This record stands for itself and need no comment.

His wife, two sons and one daughter survive him.

Dr. Edwin G. Rust

Dr. Rust, who died in Cleveland, Ohio, December 29, 1935, at the age of seventy-eight, was a graduate of the Homeopathic Medical College of Cleveland, Ohio, in 1880.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

Dr. Daniel Kerfoot Shute

Dr. Shute was born in Alexandria, Virginia, on October 22, 1858, and died in Washington, D. C., on October 21, 1935.

He obtained his early education at Columbian College, now George Washington University, and received his medical degree there in 1883, following which he began the practice of medicine in the City of Washington. He later took a postgraduate course in ophthalmology at the Royal London Ophthalmic Hospital and the University of Berlin, Germany, and confined his work to that specialty.

In 1887, Dr. Shute was made Prosector to the Chair of Anatomy of George Washington University, later becoming Clinical Professor of Ophthalmology. From 1888 to 1910 he was Professor of Anatomy and at the time of his death Emeritus Professor. During his life he was ophthalmologist to the Providence, Central Dispensary and Emergency, Columbia, and George Washington Hospitals and the Government Hospital for the Insane.

Dr. Shute was an ex-president of the Medical Society of the District of Columbia, member of the American Academy of Ophthalmology and Otolaryngology, a former president of the Society of Ophthalmologists and Otologists of Washington, Fellow of

the American College of Surgeons, member of the Anthropological and Microscopical Societies of Washington, the Association of American Anatomists, the Clinico-Pathological Society and the Virginia Medical Society.

He was a member of the Society of Cincinnati, Sons of the American Revolution, and the Cosmos Club. He was an author of many articles and publications on ophthalmology and was particularly well versed in comparative anatomy.

His widow and one daughter survive him.

Dr. Herman Bennett Slotkin

Dr. Slotkin, who died in New York City on August 1, 1936, at the age of 36 years, was a graduate of Jefferson Medical College of Philadelphia in 1925. He received the degree of M.Sc. (Med.) for graduate work in Otolaryngology at the Graduate School of Medicine of the University of Pennsylvania in 1934.

At the time of his death Dr. Slotkin was instructor in neuro-otology at the University of Pennsylvania Graduate School of Medicine, assistant neuro-otologist and assistant aural surgeon at its Graduate Hospital. He was also Assistant Otolaryngologist at the Jewish Hospital.

He was a life member of the Philadelphia County Medical Society and the Pennsylvania State Medical Society, member of the Philadelphia Laryngological Society and of the American Academy of Ophthalmology and Otolaryngology.

During the World War, Dr. Slotkin served in the Medical Corps, Chemical Warfare Division.

Dr. Slotkin was held in high regard in the clinical service at the Graduate Hospital.

Dr. Stanley Sinclair Smith

Dr. Smith of Pittsburgh, Pennsylvania, died on December 2, 1935, at the age of sixty-one.

He was a graduate of Jefferson Medical College of Philadelphia in 1896.

Dr. Smith was professor of ophthalmology at the University of Pittsburgh School of Medicine and on the staff of the Eye and Ear Hospital, Pittsburgh; Columbia Hospital, Wilkinsburg, and the Carnegie Institute of Technology.

He served during the World War, was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

Dr. Joseph Anthony Stockler

Dr. Stockler was born April 30, 1884, in Philadelphia, Pennsylvania, and died on September 9, 1935, in Reading, Pennsylvania.

He graduated from the Medical Department of the University of Pennsylvania in 1909, and after interning at the Municipal Hospital in Philadelphia and the West Penn Hospital in Pittsburgh, took up the practice of his specialties in Reading.

At the time of his death, Dr. Stockler was Chief of the Ear, Nose and Throat Department of St. Joseph's Hospital, Reading, and Laryngologist to the State Tubercular Hospitals at Hamburg and Mt. Alto.

He was a member of the American Academy of Ophthalmology and Otolaryngology and of his local and state medical societies.

He is survived by his widow and four children.

Dr. John Joseph Thomson

Dr. Thomson was born in Toronto, Canada, on January 20, 1879, and died on November 13, 1935, at Mount Vernon, New York.

After graduating from LaSalle Institute of Toronto, he received his medical degree from Toronto University in 1902, followed by a period of postgraduate work at the Manhattan Eye, Ear and Throat Hospital. He began the practice of his specialties in New York City in 1905, later moving to Mount Vernon.

At the time of his death Dr. Thomson was chief of the ear, nose and throat department of the Lawrence Hospital, Bronxville, chief of the ear, nose and throat department of the Mount Vernon Hospital, and was president of its medical board from 1915 to 1924.

He was a member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Society, the American Otological Society, Fellow of the American College of Surgeons, and member of his local and state societies.

He is survived by his widow and one brother.

Dr. William S. Tomlin

Dr. Tomlin was born on January 10, 1869, at Mount Zion, Kentucky, and died January 27, 1936, in Indianapolis, Indiana.

He attended the Indiana State Normal School at Terre Haute, Ohio Medical College in Cincinnati, and received his medical degree from the University of Louisville, Kentucky.

Dr. Tomlin began the practice of medicine in 1895 in Indianapolis, and in 1909, following postgraduate work in New York, Chicago, London, Vienna, and Berlin, limited his practice to the ear, nose and throat. He was on the staffs of St. Vincent's Methodist Episcopal, St. Francis, and Indianapolis City Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, held membership in his state and local medical societies, having been president in 1921 of the Indianapolis Academy of Ophthalmology and Otolaryngology.

He is survived by his widow and one sister.

Dr. Roy Seymour Watson

Dr. Watson was born in Breckenridge, Michigan, on December 15, 1881, and died in Saginaw, Michigan, November 27, 1935.

Receiving his medical degree from Rush Medical College in Chicago, Dr. Watson settled in Breckenridge, and limited his practice to the ear, nose and throat in 1911, after some years of general practice. He was chief of the Department of Otolaryngology of the Saginaw General Hospital.

Dr. Watson was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of his local and state medical societies.

During the World War, he was a Captain in the Medical Corps stationed at the base hospital at Camp Dodge, Des Moines, Iowa.

He is survived by his wife, mother and a sister.

Dr. William Hamlin Wilder

Dr. Wilder was born in Covington, Kentucky, on December 16, 1860, and died in Chicago, Illinois, on September 24, 1935.

He received his early education at Farmers College, College Hill, Ohio, B.A. degee at Belmont College in 1878, and his med-

ical degree from the Medical College of Ohio, Cincinnati, in 1884. He also pursued postgraduate study in the University of Göttingen in 1889 and the University of Vienna in 1890.

After several years of general practice in Cincinnati, Dr. Wilder moved to Chicago in 1892, and in 1907 became Professor of Ophthalmology at Rush Medical College, being Emeritus Professor at the time of his death. He served as honorary surgeon to the Illinois Eye and Ear Infirmary and as ophthalmologist to the Presbyterian Hospital.

Dr. Wilder was a member of the American Academy of Ophthalmology and Otolaryngology, and its president in 1931; a member of the American Ophthalmological Society, and its president in 1918; member and vice-president of the Illinois Society for the Prevention of Blindness; Chairman of the Section on Ophthalmology of the American Medical Association in 1907-1908, and member of the House of Delegates for many years; and he was also a Fellow of the American College of Surgeons. He was awarded the Leslie Dana Gold Medal by the St. Louis Society for the Prevention of Blindness in 1935, for outstanding contributions in the preservation of sight. Dr. Wilder was instrumental in the establishment of the American Board of Ophthalmology, being one of its charter members and was its secretary for many years.

He was a Major in the Medical Reserve Corps during the World War.

One feels that a personal touch should be added in recording the life of this splendid man. From his associates we learn that though somewhat austere in his early life, he gradually mellowed with time and grew to be a most patient and wise counsellor of the younger men. However trivial the work he was always meticulous and particularly painstaking in his surgery.

Dr. Wilder was fond of outdoor sports, enjoyed golf, duck hunting and horseback riding when time permitted. His attendance at medical meetings was almost a religious duty. His discussions were few but valuable and though not a research man he was an accurate clinician. The plan of the Chicago Ophthalmological Society for the Wilder Memorial Fund in the establishment of a biennial lectureship in Ophthalmology is but another testimonial of his value.

His widow, one daughter and one son survive him.

Dr. Harry Ralph Wormley

Dr. Wormley of Rockford, Illinois, who died suddenly of heart disease on November 20, 1935, while on a hunting trip in Iowa, was a graduate of the Rush Medical College, Chicago, in 1906.

At the time of his death, Dr. Wormley was on the staff of the Rockford Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

* * *

There being no objection, the report of the Historian was placed on file.

REPORT OF THE AMERICAN BOARD OF OPHTHALMOLOGY

Dr. Goar gave the following report:

As the retiring member of the delegation of the Section on Ophthalmology to the American Board of Ophthalmology, I beg to submit herewith a brief report.

The American Board of Ophthalmology has held in all, fifty examinations; 1,316 certificates have been issued. The geographical distribution is as follows:

New England States	94
Eastern States	419
Southern States	91
Middle Western States	428
Western States	151
Possessions	2
Foreign	27

132 of our diplomates have died

The increasing influence and importance of the work of the American Board of Ophthalmology is attested by the fact that forty teaching institutions and hospitals require the Board certificate for staff appointments or promotions.

The Board certificate is also required as a prerequisite for membership in ophthalmology in 25 national, state and local ophthalmological societies.

American ophthalmologists should take pride in the fact that the American Board of Ophthalmology was a pioneer in the movement to establish special examining boards. At the present time there have been formed, or are in the process of forming, twelve such Boards.

Dr. John Green was appointed Secretary to succeed Dr. Wilder.

Respectfully yours,

S. J. BEACH

There being no objection, the report of the Committee was placed on file as read.

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

During the current year the American Board of Otolaryngology has held two examinations; one in Kansas City, Missouri, May 9th, at which time thirty-four otolaryngologists were certified and one in New York City, September 25th and 26th, when 103 were granted certificates. Since the inception of the Board in 1924, thirty-two examinations have been given in twenty-five different cities. To date, 2,514 otolaryngologists have been certified, of which number ninety-nine are deceased.

REPORT OF THE AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

Dr. Charles A. Bahn read the report as follows:

The following members of a subcommittee have been appointed to serve on a National Committee of Motor Anomalies and Orthoptic Training: Dr. Conrad Berens, Dr. Charles A. Bahn, and Dr. Thomas Allen.

The following American instruments have been found of use:

1. Orthoptic Training; Stereo-Orthopter; Rotoscope.
2. In Reading Difficulties: Ophthalmograph; Metronomoscope.

Among the foreign instruments believed worthy of consideration by American ophthalmologists are:

Emile Sydow, Berlin: 1. Hand Ophthalmoscope. Dr. Comberg: direct and indirect methods. 2. Hand keratometer, Dr. Springer; attaches to flashlight. 3. Loupe for the color blind.

Oculus, Berlin: 1. Vertex lensometer; small, compact, inexpensive.

Carl Zeiss: 1. Hand ophthalmoscope; direct and indirect methods; Morton lens system. 2. Ophthalmometer. 3. Perimeter, Dr. Maggiori.

Emile Busch, Rathenow: 1. Pocket ophthalmoscope, reflex-free. 2. Vertex lensometer; small, compact and inexpensive.

Haag and Streit, Bern, Switzerland: 1. Slit light; resembles Koeppe type. Johan Greishaber, Scharfhausen, Switzerland: Complete line of ophthalmic instruments in both case and stainless steel; instruments used in retinal detachment by Vogt and Gonin. Theodore Hamlin, London: 1. McKie Reid spinal spectacles, for reading in bed. 2. Sheila Mayou strabismuscope; a new orthoptic training instrument incorporating coordination of hand and eye.

Laws for the regulation and licensing of dispensing opticians are approved in principle.

The following subcommittee has been appointed for the study of the principles of lighting requirements: Cowan, Lancaster, and Berens.

Respectfully submitted,

AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

There being no objection, the report of the Committee on Optics and Visual Physiology was accepted.

REPORT OF THE NATIONAL OTOLARYNGOLOGICAL LIBRARY

DR. BURT R. SHURLY: The National Otolaryngological Library Committee has carried on a program of assisting the Surgeon-General's office in trying to establish a better arrangement for the literature that is accumulating in the Surgeon-General's

library. The Registry, of which Dr. Joseph Beck will speak, has really been a part of the movement in trying to register and supply a general collection of those things that have to do with the library and the museum.

The Surgeon-General's library should have a new and larger building. It is hoped that the members of the Academy will be interested in speaking of this to their Congressmen and to their Senators, because that appropriation, according to Surgeon-General Reynolds, would at once take care of all the library's facilities, of all the specialties in the country and would give an opportunity for our museum and library to be increased and made more available for work in reference and in general preservation of specimens, and so forth.

It is hoped that the members will take an active interest in endeavoring to get their appropriation for a new and larger Surgeon-General's library.

There being no objection, the report of the Committee was adopted.

REPORT OF THE COMMITTEE ON DEAFNESS PREVENTION AND AMELIORATION

Your Committee again reports continued progress. Its members have worked to promote a better understanding and greater activity in the field of deafness prevention through numerous addresses and published papers on subjects pertaining to this field; and in helping to stimulate interest and successful effort in the formation of Committees on Deafness Prevention and Amelioration in State Medical Associations.

They have made initial efforts to promote the securing of effective state legislation which will make obligatory the expansion of the school health program so as to include the regular, periodic testing of the hearing of all school children by strictly modern methods, and insure the needed care of those with defective hearing in a manner which will not be prejudicial to the interests of the practicing physician.

Educational exhibits have been sponsored to teach the public the need and possibilities of conserving the hearing, at which ethical, informative printed matter has been distributed to thousands of interested inquirers.

The research project at the University of Minnesota dealing with the performance characteristics of electric hearing aids and some of the newly released audiometers has been continued with results advantageous both to the consumers and manufacturers of these products. A preliminary report was published in the July number of the Archives of Otolaryngology.

Because of its bearing on ear diseases the improved film on the Hygiene of Swimming, produced by one of our members, Dr. H. Marshall Taylor, Chairman of the American Medical Association Committee on the Hygiene of Swimming, has been exhibited before many interested groups of physicians, nurses, educators, Boy Scout Leaders and Boy Scouts.

A definite plan for more comprehensive and effective work as justified by the previous studies of your Committee is now under serious consideration. The greatest obstacle to rapid progress has been the lack among our profession of specific knowledge of the fundamental needs and possibilities of attainment in work in this field of preventive medicine. Their increased interest shows that this obstacle is being gradually overcome.

The Academy will be represented at the coming International session of the Inter-State Postgraduate Medical Association of North America, October 12th to 16th in St. Paul, Minnesota, by an exhibit of your committee.

In view of the large amount of work remaining to be done in this field it is our recommendation that the committee be continued and the grant to help meet the costs of its activities, be renewed.

Respectfully submitted,

John F. Barnhill	Austin A. Hayden
Gordon Berry	Frederick T. Hill
T. E. Carmody	Burt R. Shurly
E. H. Cary	H. A. Smith*
G. M. Coates	H. Marshall Taylor
H. V. Dutrow	Claude T. Uren
A. C. Furstenberg	

Horace Newhart, *Chairman*

* Elected September, 1936.

REPORT OF THE COMMITTEE ON PUBLIC RELATIONS

Dr. Ralph A. Fenton read the following report:

Continuing the policy of public instruction heretofore authorized by the Council, your Committee on Public Relations secured abstracts and prepared and sent out releases to the national press associations and science editors of nationally circulated periodicals to the number of 31. Through the courtesy of the New York Academy of Medicine, four broadcasts were delivered by Sir John Parsons on "The Preservation of Sight," by Dr. Dean on "The Eye and Ear Specialist and the Public Health," by Dr. Shurly on "Prevention of Deafness in the School Child," and by Dr. Mosher on "Sore Throat." Twenty-four special releases were prepared in New York during the meeting at the request of New York men because of things that came up in the meeting since we have reached New York.

The services of Dr. C. L. Levenson of New York, an experienced newspaper man, were arranged for last January, and he has been of inestimable value in preserving cordial relations with the press, while we have limited the output of special news stories as far as seems possible under conditions obtaining in this community. Names of local physicians were, as far as possible, excluded from publicity, and your committee desires to express its very sincere appreciation of the cooperation of Drs. Wheeler and Jones, as well as the editors and special writers for national and local press associations, local newspapers, and periodicals reporting on scientific medicine.

Continuation of current policies has been recommended by your committee.

Respectfully submitted,

ERLING W. HANSEN
RALPH A. FENTON
Chairman

There being no objection, the report of the Committee on Public Relations was adopted.

REPORT OF THE COMMITTEE ON OPHTHALMIC PATHOLOGY

Dr. Harry S. Gradle stated that Major Cornell would present the technical report. Major Cornell then stated that due to the illness of the Curator at the Museum, he was representing the Museum at the session and read the following report:

REGISTRY OF OPHTHALMIC PATHOLOGY

Advisory Committee

Dr. Harry S. Gradle ..... Chicago
Dr. F. H. Verhoeff ..... Boston
Dr. Jonas S. Friedenwald ..... Baltimore
Dr. Georgiana D. Theobald ..... Chicago
Curator, Army Medical Museum, Washington, D. C., Registrar

The numerical status of the Registry of Ophthalmic Pathology on December 31, 1935, was as follows:

TABLE I

YEAR	NO. OF ACCESSIONS
1919-1931	3,910*
1932	311
1933	317
1934	309
1935	415
Total	5,262

* Includes 1,981 illustrations, models, and eye specimens which were donated to the registry from other collections of Ophthalmic Pathology, notably that of the late Dr. James Moores Ball of St. Louis, Missouri.

The marked increase in the number of accessions for 1935 is due to a greater number of eye specimens sent in for examination and diagnosis. It is through this service that the opthalmic collection is gradually being built up in size and importance. As the value of this collection for study and research is becoming better known to ophthalmologists throughout the United States, an increasing number of requests are received for study material.

The following tables appended to this report are the same as those for previous years brought up to date by the addition of the news cases and follow-up data received during the year:

TABLE II

Classification by Histologic Type of 319 Primary Melanotic Tumors of the Eye, Showing Age, Distribution, Location, Degree of Pigmentation and Outcome after Enucleation, December 31, 1935

TYPE		AGE AT OPERATION						LOCATION			PIGMENT			STATUS AFTER OPERATION							DEATHS		
	Total Cases	20 to 39	40 to 49	50 to 59	60 to 69	70 and over	Not known	Uvea	Limbus	Orbital extension from uvea	Slight	Moderate	Abundant	3 years or less		3 to 5 years		5 years and over		Patient lost	Tumor	Other	Unknown
														L	D	L	D	L	D				
Spindle A	40	3	11	12	8	4	2	40	0	7	19	11	10	12	4	8	0	8	1	7	0	4	1
Spindle B	64	13	9	19	21	1	1	63	1	14	34	25	5	27	5	15	3	5	2	7	3	6	1
Epithelioid	25	7	9	3	5	1	0	13	12	4	6	14	5	10	5	4	0	2	0	4	4	1	0
Fascicular	17	5	3	4	2	3	0	17	0	8	10	4	3	4	7	4	0	1	0	1	3	3	1
Mixed	173	29	30	48	39	23	4	165	8	57	29	68	76	36	45	11	10	22	9	40	52	10	2
TOTAL	319	57	62	86	75	32	7	298	21	90	98	122	99	89	66	42	13	38	12	59	62	24	5

L = Living D = Dead

TABLE III

Classification by Histologic Type and Fiber Content of 135 Cases of Melanoma of the Choroid, December 31, 1935

FIBER CONTENT	SPINDLE A					SPINDLE B					EPITHELIOID					FASCICULAR					MIXED					LIVING		DEAD		TOTAL CASES	% DEAD	AVERAGE POST-OPERATIVE DURATION IN FATAL CASES		
	Yrs. Living			Total		Yrs. Living			Total		Yrs. Living			Total		Yrs. Living			Total		Yrs. Living			Total		Tot.	Ext.	Tot.	Ext.					
	1-3	3-5	5+	L	D	1-3	3-5	5+	L	D	1-3	3-5	5+	L	D	1-3	3-5	5+	L	D	1-3	3-5	5+	L	D									
1. No fibers among tumor cells	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	2	2	0	0	2	2	3	3	4	0	7	57+		1-4/12	
2. Partially fibered																																		
a. Less than 50% fibers	1	2	1	4	0	8	2	1	11	3	1	0	0	1	1	0	1	0	1	0	6	3	3	12	19	29	14	23	8	52	44+	33+	1-9/12	2-3/12
b. About 50% fibers	1	1	2	4	0	3	7	0	10	1	0	1	0	1	2	1	1	0	2	1	5	2	3	10	11	27	10	15	7	42	35+		1-11/12	
c. More than 50% fibers	0	1	2	3	0	2	3	1	6	0	0	0	0	0	0	0	1	0	1	0	1	2	4	7	5	17	6	5	4	22	22+		3-3/12	
3. Fibers throughout all areas	3	1	2	6	0	0	2	3	5	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	12	1	0	0	12	0		0	
TOTAL	5	5	7	17	0	13	14	5	32	4	1	1	0	2	3	1	4	0	5	3	14	7	11	32	37	88	34	47	19	135	35+		2	

L = Living D = Dead Ext. = Extrabulbar extension from choroid tumor.

TABLE IV

Classification by Histologic Type and Fiber Content of 54 Cases of Melanoma of the Choroid followed 5 years or more December 31, 1935

Fiber Content	Spindle A		Spindle B		Epithelioid		Fascicular		Mixed L	Mixed D	Living Total	Living Ext.	Dead Total	Dead Ext.	Total Cases	% Dead	
1. No fibers among tumor cells	0	0	0	0	0	0	0	2	0	1	0	0	3	0	3	100	
2. Partially Fibered																	
a. Less than 50% fibers	1	0	1	1	0	1	0	0	3	12	5	2	14	4	19	73+	}
b. About 50% fibers	2	0	0	0	0	1	0	1	3	9	5	1	11	4	16	68+	} 57+
c. More than 50% fibers	2	0	1	0	0	0	0	0	4	3	7	3	3	2	10	30	}
3. Fibers throughout all areas	2	0	3	0	0	0	0	0	1	0	6	0	0	0	6	0	
TOTAL	7	0	5	1	0	2	0	3	11	25	23	6	31	10	54	57+	

TABLE V

Status of 115 Cases of Retinoblastoma in the Registry of Ophthalmic Pathology, Army Medical Museum, on December 31, 1935

Age at Operation	Male	Female	Sex unknown	Unilateral	Bilateral	Post-Oper. Duration Living Years —1	1-2	2-3	3-4	4-5	5+	Total Living	Dead	Status unknown	Total
— 1	4	8	0	8	4	4	1	2	0	2	2	11	1	0	12
1-2	8	10	0	10	8	0	2	2	2	2	4	12	3	3	18
2-3	19	9	0	22	6	2	1	1	0	0	5	9	16	3	28
3-4	19	7	1	20	7	1	3	1	1	0	2	8	14	5	27
4-5	5	5	1	9	2	0	1	0	0	1	2	4	5	2	11
5-6	2	2	0	4	0	1	0	0	0	0	0	1	3	0	4
6-11	3	4	0	5	2	0	1	2	0	0	0	3	2	2	7
Age unknown	2	0	6	8	0	0	0	0	0	0	0	0	0	8	8
TOTAL	62	45	8	86	29	8	9	8	3	5	15	48	44	23	115

TABLE VI

Comparative Status of 92 Cases of Retinoblastoma from the Standpoint of Extension and Bilateral Involvement, December 31, 1935

AGE (years) AT OPERATION	UNILATERAL Ext.+ L	Ext.+ D	Ext.— L	Ext.— D	Total L	Total D	BILATERAL Ext.+ L	Ext.+ D	Ext.— L	Ext.— D	Total L	Total D
— 1	3	0	5	0	8	0	1	0	2	1	3	1
1-2	1	2	5	0	6	2	3	1	3	0	6	1
2-3	4	9	4	1	8	10	0	6	1	0	1	6
3-4	3	9	4	2	7	11	1	3	0	0	1	3
4-5	1	5	1	0	2	5	0	0	2	0	2	0
5-6	0	3	1	0	1	3	0	0	0	0	0	0
6-11	1	2	2	0	3	2	0	0	0	0	0	0
TOTAL	13	30	22	3	35	33	5	10	8	1	13	11

Ext.+ L = 18 cases
Ext.+ D = 40 cases
L — Living

Ext.— L = 30 cases
Ext.— D = 4 cases
D — Dead

Ext.— Without extrabulbar extension of tumor
Ext.+ Extrabulbar extension of tumor

TABLE VII

Tabulation by Mode of Extension of 92 cases of Retinoblastoma of which the present status is known, December 31, 1935

EXTENSION				POST-OPERATIVE DURATION (YEARS)								
Optic Nerve	Choroid	Ciliary Body	Sclera	1—	2—	3—	4—	5—	5+	Total Living	Dead	Total
—	—	—	—	6	6	1	1	2	10	26	4	30
+	+	+	+	0	0	0	0	1	0	1	15	16
+	+	—	—	3	0	0	3	0	0	6	5	11
+	+	—	+	0	0	1	0	0	1	2	6	8
+	+	+	—	0	0	0	0	0	0	0	6	6
+	—	—	—	0	1	2	0	1	3	7	5	12
—	+	—	—	0	1	1	0	0	2	4	0	4
+	—	+	+	0	0	0	0	0	0	0	1	1
+	—	—	+	1	0	1	0	0	0	2	1	3
—	—	+	—	0	0	1	0	0	0	1	0	1
TOTAL				10	8	7	4	4	16	49	43	92

TABLE VIII

Tabulation of 89 Cases of Epithelial Tumors of the Ocular Adnexa, December 31, 1935

	AGE AT OPERATION							ORIGIN			LIVING							DEAD			
											YEARS POST-OPERATIVE										
	—20	20-39	40-49	50-59	60-69	70+	Unknown	Lid	Conjunctiva	Cornea	—1	1-2	2-3	3-4	4-5	5+	TOTAL LIVING	Tumor	Others	Unknown	No report since operation
Papilloma	2	5	1	2	2	1	0	4	7	2	4	1	2	1	1	1	10	0	0	0	3
Carcinoma	2	10	10	19	19	14	6	38	40	2	7	13	6	4	6	8	44	7	4	2	23
TOTAL	4	15	11	21	21	15	6	42	47	4	11	14	8	5	7	9	54	7	4	2	26

The following articles based on original research, the results of which are shown in Tables III and IV, were published in 1935:

An Improved Technique for Silver Impregnation of Reticulum Fibers. Helenor Campbell Wilder. Am. J. of Path., xi:817-819, Sept. 1935.

Melanoma of the Choroid: The Prognostic Significance of Argyrophil Fibers. G. R. Callender, M.D., and Helenor Campbell Wilder. Am. J. Cancer, vol. xxv, No. 2, Oct. 1935.

With respect to these two articles, may I say one word in explanation, Mr. President? You are all familiar with Colonel Callender's classification by cell type of melanomas of the eye. The non-malignancy of the spindle A group is an established fact. However, no definite conclusions were arrived at from histological study of the mixed group.

Mrs. Wilder, in trying various silver stains, observed that certain of these tumors contained more argyrophil fibers than others. Following that, the tumors were reclassified on the basis of the relation of their fiber content to the clinical outcome. This classification has proved a definite aid to Callender's original classification, particularly in tumors combining both spindle and epithelioid types. This material is all given in detail in the tables which are appended to the report.

No changes were made in the loan sets of pathology and histology during 1935 but a number of slides were cut and stained with a view to substitution during the coming year. New history blanks which provide more information with less effort on the part of the contributor have been devised and printed.

A collection of lantern slides illustrating the normal anatomy of the eye was assembled and has proved to be so satisfactory that repeated requests have been received for its use. This is another evidence of the desire on the part of the Army Medical Museum to cooperate with the Academy to the fullest extent. Because the 12 large loan sets of ophthalmic pathology are inadequate to supply the demand, it is planned to prepare for circulation 20 sets of 25 slides each. These are to be accompanied by catalogues containing photomicrographs and descriptive and historical material. They are particularly for use in the Academy's courses in histopathology and in preparation for Board examination.

REGISTRY OF OTOLARYNGIC PATHOLOGY

Advisory Committee

Dr. Joseph C. Beck .....Chicago
Dr. Frank J. Novak, Jr. .....Chicago
Dr. LeRoy A. Schall .....Boston
Dr. Herman Z. Seminov .....Los Angeles
Curator, Army Medical Museum, Washington, D. C., Registrar

In anticipation of the organization of this registry of Otolaryngic Pathology, Dr. Harris P. Mosher contributed paraffin blocks from 525 cases in his laboratory. These have all been recut and slides prepared for the collection. Representative cases for loan sets of otolaryngic histology and pathology, similar to the ophthalmic sets, have been selected from this collection, from recent cases contributed to the Registry and from the Museum files. Approximately fifty of these have been cut, stained and photographed. It is planned to prepare twenty sets of a hundred slides each with catalogues containing descriptive material and photomicrographs. History blanks have been devised and printed for registering cases of otolaryngic pathology. All tumors are being carded and followup blanks will be sent to the contributors of tumor cases each January. The total number of cases in the Registry on December 31, 1935, was 550.

The financial support furnished by the Academy to the Registries of Ophthalmic and Otolaryngic Pathology has been a substantial aid in carrying on the work of these registries.

There being no objection, the report was placed on file.

REPORT OF THE COMMITTEE ON OTOLARYNGIC PATHOLOGY

DR. JOSEPH C. BECK: Major Cornell has given you the report somewhat in detail. I wish to talk about my recent visit to the Army Medical Museum and the hour that I spent with the Surgeon General discussing the ultimate disposal of the National Library of Otolaryngology. There is serious need for a new building to house the Army Medical Museum and Library and it is important that the committee should keep alive interest in the Registry and the housing of its material. The committee has had no occasion

for a meeting but its members have talked over this subject informally. We are doing all we can to see that the new building is established. Colonel Jones, who has taken on the duties of librarian succeeding Colonel Hume, is very much interested in our Otolaryngic library.

Dr. Shurly is correct in his statement regarding what members of the Academy can do in their own localities towards obtaining a proper Army Medical Museum and Library building. It would be a terrible thing to lose, by fire, these great books and this irreplaceable pathologic material.

I had the pleasure of seeing the work that is being done for the Registry of Otolaryngology at the Army Medical Museum. Reports are inadequate, and those who are interested should go there and see for themselves what the Army is doing for the Societies. It is impossible for the museum staff to perform, within their regular hours, the vast amount of work required by the registries. The money that is contributed by these two Boards is the only outside financial support, and, because it is insufficient to pay the salary of an additional assistant, it is used to compensate the civilian members of the staff for their overtime work.

I want to mention something here that came up the other night at the meeting where we discussed the Registry. Matters were discussed in Oto- and Ophthalmologic Pathology. It was stated that there is enough material there so that a man could be sent there and do a piece of work either in Ophthalmic or Otolaryngologic Pathology that would be a great credit. It is a pity that so much material, accumulated histories, photographs, and so forth, is not being used for development of some fellow who would be sponsored not by the society but by individual supporters.

We are very proud of this Registry, being so young, and it has already made fairly good headway. We will have to keep on working.

The office in Omaha should give us greater aid in circularizing the Otolaryngologic Pathology Registry. I am going to keep at it until we do get it. We need help and the assistance of the men throughout the country to send preparations there. They can use some of their preparations in their own institutions, but they always have a part of the block left, just as you heard that Dr. Mosher sent a great many blocks there. These paraffin blocks should be sent, providing there is a good history accompanying them.

There being no objection, the report was adopted.

REPORT OF THE SENIOR MEMBER OF COUNCIL

Dr. Burt R. Shurly read the report as follows:

Research Appropriations:

International Trachoma Investigation	$ 100
Surgeon-General — Registry of Pathology	1000
Committee on Prevention and Amelioration of Deafness	350
Dr. Lurie, Boston	400
Drs. Larsell and Fenton, Portland, Ore.	400

The Transactions for 1936 are to be dedicated to General Sir Herbert Birkett, of Montreal, Canada.

The question of continuing the Scientific Exhibit has been left to the discretion of the Program Committee for each year. The Secretarial Group has been instructed to include the Scientific Exhibit as a part of their management portfolio.

The Council desires to express sincere appreciation to Doctors Kopetzky and Lloyd, their committee, and to all those who, through exhibits, have made of this activity an outstanding factor in the New York Convention.

The Council desires at this time to express appreciation to all the New York Committees for the very splendid cooperation in consummating the plans for the convention.

Dr. Harry A. Smith, of Whittier, California, has been added to the Committee on the Prevention and Amelioration of Deafness.

The report of the Council was adopted without objection.

There was no unfinished business and no new business on the Secretary's table.

Election of Members

DR. WHERRY: Mr. President, the Council has considered and desires to nominate the name of Sir John Herbert Parsons for Honorary Membership.

The report of the Council regarding the Honorary Membership was carried by a standing vote.

DR. WHERRY: The Council desires at this time to pay their sincere respects to the late Arthur B. Duel, and I desire to nominate for Honorary Membership the late Arthur B. Duel.

There being no objection, the report of the Council was accepted by a standing vote.

DR. WHERRY: Pursuant to our by-laws, the following members have been elevated from Senior to Life Members:

C. S. Beebe, Milwaukee, Wisconsin
R. W. Bledsoe, Covington, Kentucky
F. E. Brawley, Chicago, Illinois
Frank E. Burch, St. Paul, Minnesota
Lee W. Dean, St. Louis, Missouri
G. C. Hall, Louisville, Kentucky
W. E. Hopkins, Los Angeles, California
Samuel Iglauer, Cincinnati, Ohio
Charles A. Ringle, Greeley, Colorado
F. McK. Ruby, Union City, Indiana
E. H. Thompson, Cincinnati, Ohio
Helen F. Upham, Asbury Park, New Jersey

The following to Senior Membership:

Francis J. Arnold, Burlington, Vermont
H. T. Aynesworth, Waco, Texas
Fred W. Bailey, Cedar Rapids, Iowa
Arthur J. Bedell, Albany, New York
W. F. Boiler, Iowa City, Iowa
G. H. Boyce, Iron Mountain, Michigan
W. Howard Brown, Edmonton, Canada
Edwin Cobb, Marshalltown, Iowa
Glendon E. Curry, Pittsburgh, Pennsylvania
C. H. Dewey, San Francisco, California
H. H. Glosser, Buffalo, New York
Perry Goldsmith, Toronto, Canada
Fred E. Grant, Kalamazoo, Michigan
Harold M. Hays, New York, N. Y.
E. E. Holt, Portland, Maine
Douglas P. A. Jacoby, Newport, Rhode Island
W. H. Johnston, Santa Barbara, California
H. G. Kline, Syracuse, New York
Oscar H. Kraft, Chicago, Illinois

H. B. Lemere, Beverly Hills, California
George W. Mackenzie, Philadelphia, Pennsylvania
G. Henry Mundt, Chicago, Illinois
S. L. Olsho, Philadelphia, Pennsylvania
Harry E. Peterman, Baltimore, Maryland
E. H. Porter, Tiffin, Ohio
Fred J. Pratt, Minneapolis, Minnesota
H. B. Searcy, Tuscaloosa, Alabama
Elbert S. Sherman, Newark, New Jersey
Victor C. Smith, New Orleans, Louisiana
Fred Stauffer, Salt Lake City, Utah
C. B. Voigt, Mattoon, Illinois
Carl B. Wagner, Chicago, Illinois

Under the authority of the Council, I desire to nominate the following candidates for membership, whose credentials have been carefully examined and approved:

Adelman, Benjamin Barnet, 190 Clinton Ave., Newark, N. J., Op.
Albers, Edward Charles, 44 Main Street, Champaign, Ill., Op.
Alexander, Charles Sanford, Medical Arts Bldg., Houston, Texas, Op.
Alger, L. James, Grand Forks, N. D., ALR
Allen, Cary David, U. S. Naval Hospital, Newport, R. I., ALR
Allen, Donald Gray, 1021 Prospect Ave., Cleveland, Ohio, Op.
Allis, Paul Mitten, 6 N. Brown St., Lewistown, Pa., ALR
Andrews, Albert H., Jr., 25 E. Washington St., Chicago, Ill., ALR
Appelbaum, Alfred, 115 East 61st St., New York, N. Y., Op.
Atkinson, John Hedley, 168-70 Sterling St., Watertown, N. Y., ALR
Ball, Simon, 311 S. 20th St., Philadelphia, Pa., ALR
Barlow, Aaron, 1307 Spruce St., Philadelphia, Pa., Op.
Beavis, James Olin, Fidelity Bldg., Dayton, Ohio, ALR
Bell, Julius W., 307 East 17th St., New York, N. Y., ALR
Bishop, Frederick J., Medical Arts Bldg., Scranton, Pa., ALR
Blankfeld, Nathan, 1506 Ocean Ave., Brooklyn, N. Y., ALR
Boies, Lawrence Randall, Medical Arts Bldg., Minneapolis, Minn., ALR
Bowers, Wesley Creveling, 17 East 61st St., New York, N. Y., ALR
Brandenburg, Nora B., 25 E. Washington St., Chicago, Ill., ALR
Brannin, Daniel, Medical Arts Bldg., Dallas, Texas, Op.

Braun, Jacob, 270 West End Ave., New York, N. Y., ALR
Brighton, George Renfrew, 580 Park Ave., New York, N. Y., ALR
Briglia, Frank Joseph, 703 Wharton St., Philadelphia, Pa., ALR
Broones, Otto, 302 West 87th St., New York, N. Y., ALR
Buckley, Robert, 50 East 63rd St., New York, N. Y., ALR
Buffum, Edward S., 40 W. North St., Buffalo, N. Y., ALR
Burchfield, George Winston, 309 Court St., Maryville, Tenn., ALR
Calcote, Royal Jackson, Donaghey Bldg., Little Rock, Ark., Op.
Calvert, Raymond R., Schultz Bldg., LaFayette, Ind., Op.
Campbell, Sherburne, 147 West Main St., Meriden, Conn., Op.
Campbell, William Edward, Jr., Medical Arts Bldg., Atlanta, Ga., Op.
Champion, Albert Nicholas, Medical Arts Bldg., San Antonio, Texas, ALR
Chapman, Katharine Howe, 122 S. Michigan Blvd., Chicago, Ill., Op.
Cheney, Ralph Edwin, 105 South 7th St., Salina, Kansas, Op.
Chisholm, Julian Ford, Jr., 512 Abercorn St., Savannah, Ga., Op.
Cinelli, Albert A., 1021 Park Ave., New York, N. Y., ALR
Cohen, Louis Benjamin, 1521 Arrott St., Philadelphia, Pa., Op.
Constantine, K. W., Medical Arts Bldg., Birmingham, Ala., Op.
Cook, Raymond Clio, Donaghey Bldg., Little Rock, Ark., Op.
Corboy, Philip M., 8 Monroe St., Valparaiso, Indiana, Op.
Cottle, Maurice H., 30 N. Michigan, Chicago, Ill., ALR
Cox, Charles Lynwood, 150 Baronne St., New Orleans, La., ALR
Curtin, John F., Medical Arts Bldg., Minneapolis, Minn., ALR
Davies, Windsor S., Eaton Tower, Detroit, Mich., Op.
Davis, Oscar, U. S. Naval Hospital, Philadelphia, Pa., OALR
Davison, Francis Wells, Geisinger Hospital, Danville, Pa., ALR
Dickinson, William Gilford, State Tower, Syracuse, N. Y., Op.
Douglas, Edward M., 8785 153rd St., Jamaica, L. I., N .Y., Op.
Downing, James Arthur, Bankers Trust Bldg., Des Moines, Iowa, ALR
Druss, Joseph George, 1235 Park Ave., New York, N. Y., ALR
Dumas, Edward DeVan, Medical Arts Bldg., San Antonio, Texas, ALR
Dunn, Louis Simon, 255 S. 17th St., Philadelphia, Pa., ALR
Dwyer, James Garfield, 300 Park Ave., New York, N. Y., ALR
Earl, Harry David, 479 Sixth St., San Pedro, Calif., ALR
England, Welch, 621 Market St., Parkersburg, W. Va., ALR
Fabricant, Noah D., 185 N. Wabash Ave., Chicago, Ill., ALR
Fast, Ralph Bryan, Am. Nat'l Bank Bldg., Kalamazoo, Mich., ALR

Faulkner, E. Ross, 101 East 58th St., New York, N. Y., ALR
Feldman, William, 2021 Grand Concourse, New York, N. Y., ALR
Foster, George Clark, 807 Broadway, Fargo, N. D., ALR
Frazee, John Richard, 311 Beacon St., Boston, Mass., ALR
Fred, Gus Bernard, 475 Commonwealth Ave., Boston, Mass., ALR
Friesner, Isidore, 36 East 73rd St., New York, N. Y., A
Furman, Martin A., 142 West 87th St., New York, N. Y., ALR
Garlick, George Burroughs, 144 Golden Hill St., Bridgeport, Conn., ALR
Garrick, Thomas Joseph, 40 East 61st St., New York, N. Y., ALR
Gilbert, Joseph George, 50 Plaza St., Brooklyn, N. Y., ALR
Gill, Earl King, Medical-Professional Bldg., Corpus Christi, Texas, ALR
Gilmore, Milton Alfred, 901 Main St., Hopkinsville, Ky., Op.
Givner, Isadore, 108 East 66th St., New York, N. Y., Op.
Golden, Harold M., 25 E. Washington St., Chicago, Ill., ALR
Goldstein, Henry Zuckerman, 31 Lincoln Park, Newark, N. J., ALR
Gonne, William S., David Whitney Bldg., Detroit, Mich., ALR
Goodale, Robert Lincoln, 258 Beacon St., Boston, Mass., ALR
Goodman, Edgar Leonard, 1801 Eye St., N.W., Washington, D. C., Op.
Gordon, Benjamin Lee, 1616 Pacific Ave., Atlantic City, N. J., Op.
Gorman, James Raymond, 725 Church St., Lynchburg, Va., ALR
Graef, Fred W., 80 East 77th St., New York, N. Y., ALR
Greene, James Sonnett, 126 East 30th St., New York, N. Y., ALR
Griffith, Charles M., 140 East 54th St., New York, N. Y., ALR
Hardy, Walter Baber, 2121 Highland Ave., Birmingham, Ala., ALR
Havens, Fred Z., Mayo Clinic, Rochester, Minn., ALR
Hayes, Robert Thomas, 42 Coburg St., St. John's, N. B., Canada, ALR
Hedlund, Gerald Oliver, National Bank Bldg., Painesville, Ohio, ALR
Hertz, William Jacob, 125 N. 8th St., Allentown, Pa., ALR
Hirsch, Albert, 125 East 84th St., New York, N. Y., ALR
Hofmann, Albert Peter, 632 Vine St., Cincinnati, Ohio, ALR
Holding, Bruce Fowler, Bell Bldg., Montgomery, Ala., ALR
Holmes, Edgar Miller, 520 Commonwealth Ave., Boston, Mass., ALR
Houlahan, James M., 117 S. 2nd Ave., Mt. Vernon, N. Y., Op.

Hunt, Guy Anderson, Butler County Nat'l Bank Bldg., Butler, Pa., Op.
Huvelle, Rene H., 17 East 38th St., New York, N. Y., ALR
Hyman, Morris, Doctors Bldg., Cincinnati, Ohio, ALR
Ireland, Percy E., Medical Arts Bldg., Toronto, Ont., Can., ALR
Johnston, Hewitt, Exchange Bldg., Orlando, Fla., ALR
Jones, Fred Goodwin, 258 Genesee St., Utica, N. Y., ALR
Joyce, William M., 121 Main St., Middletown, Conn., ALR
Kapp, Henry, 50 Merrimack St., Haverhill, Mass., ALR
Keil, Peter A., 118 Pennsylvania Ave., Brooklyn, N. Y., ALR
Kennedy, William A., 372 St. Peter St., St. Paul, Minn., Op.
Kernan, John Devereux, 103 East 78th St., New York, N. Y., ALR
Kleinfeld, Louis, 17 East 84th St., New York, N. Y., ALR
Kluever, Herman Christof, University Hospital, Iowa City, Iowa, Op.
Koller, Herman Michael, Medical Arts Bldg., Minneapolis, Minn., Op.
Last, Murray A., 1123 Park Ave., New York, N. Y., Op.
Lazar, Nathan Kent, 55 E. Washington St., Chicago, Ill., Op.
Leahey, Brendan D., 9 Central Ave., Lowell, Mass., Op.
LeTellier, Forrest Shetterly, Medical Bldg., Knoxville, Tenn., Op.
Liggett, Harold, 133 E. 58th St., New York, N. Y., ALR
Lindsay, John R., 950 East 59th St., Chicago, Ill., ALR
Livshin, Norman, University Bldg., Syracuse, N. Y., ALR
Long, John C., Metropolitan Bldg., Denver, Colo., Op.
Loré, John Marion, 325 West End Ave., New York, N. Y., ALR
Macpherson, Duncan, 140 East 54th St., New York, N. Y., ALR
Malcolm, Percy Edwin D., 410 Park Ave., New York, N. Y., ALR
Maliniak, Jacques W., 1125 Park Ave., New York, N. Y., ALR
Malone, Phocian Ward, Petroleum Bldg., Big Spring, Texas, ALR
Manion Marlow W., Hume-Mansur Bldg., Indianapolis, Ind., ALR
Marshall, Don, University Hospital, Ann Arbor, Mich., Op.
Martin, Ashby Goddin, 540 Park Ave., New York, N. Y., ALR
McConkie, Willis Leslie, Carroll, Iowa, ALR
McGregor, D'Arcy, 83 Bryant St., Buffalo, N. Y., ALR
McLeod, Angus Morrison, Medical Arts Bldg., Toronto, Ont., Canada, ALR
Milligan, Roy H., 3720 Washington Blvd., St. Louis, Mo., ALR
Mishler, Donald Lloyd, Grand Island Clinic, Grand Island, Nebr., ALR
Moghtader, Mansoor, 40 East 62nd St., New York, N. Y., ALR

Moon, Seymour Boston, 200 Ninth St., Pittsburgh, Pa., Op.
Morwitz, Samuel M., 55 E. Washington St., Chicago, Ill., ALR
Moshinsky, Alexander L., 234 E. 20th St., New York, N. Y., ALR
Muldoon, Wilfrid Edward, Nix Professional Bldg., San Antonio, Texas, Op.
Murphy, Edward S., Montana Bldg., Missoula, Mont., Op.
Myers, David, 2109 S. 16th St., Philadelphia, Pa., ALR
Nail, James Brown, 1300 Eighth St., Wichita Falls, Texas, ALR
Neely, Paul T., Medical Arts Bldg., Portland, Ore., ALR
Neer, Edmonde DeWitte, 115 East 61st St., New York, N. Y., ALR
Nelson, Orville N., U. S. Veterans Hosp., Bay Pines, Fla., ALR
Nesbit, Wellwood, First Nat'l Bank, Madison, Wis., ALR
Ogden, James Cheston, Cooper Clinic, Fort Smith, Ark., Op.
Ohmart, Walter A., Republic Bldg., Denver, Colo., Op.
Page, John Randolph, 127 East 62nd St., New York, N. Y., ALR
Palmer, Bascom Willcox, Boston Bldg., Salt Lake City, Utah, Op.
Park, M. Benjamin, 360 Park Ave., Paterson, N. J., Op.
Paton, R. Townley, 33 East 68th St., New York, N. Y., Op.
Perera, Charles Allen, 70 E. 66th St., New York, N. Y., Op.
Peterson, John C., Sharp Bldg., Lincoln, Nebr., Op.
Peterson, William Garrett, 106 E. 13th St., Ada, Okla., ALR
Pischel, Dohrmann K., 490 Post St., San Francisco, Calif., Op.
Place, E. Clifford, 59 Livingston St., Brooklyn, N. Y., Op.
Poole, Wm. Aubrey, Security Trust Bldg., Lexington, Ky., ALR
Posner, Adolph, 667 Madison Ave., New York, N. Y., Op.
Powers, Harold Wayne, Mills Bldg., Topeka, Kansas, ALR
Rabbiner, Max, 358 New York Ave., Brooklyn, N. Y., ALR
Randel, Harvey Ollis, Commerce Bldg., Okmulgee, Okla., Op.
Robert, Joseph X., 170 St. George St., Toronto, Canada, ALR
Rosenberg, Joseph, 333 Linwood, Buffalo, N. Y., ALR
Rosenberger, Henry P., 221 Fifth St., Bismarck, N. D., ALR
Rosenwasser, Harry, 49 East 96th St., New York, N. Y., ALR
Ross, John, 33 East 68th St., New York, N. Y., ALR
Rubin, Nathan Samuel, 3401 Fifth Ave., Pittsburgh, Pa., Op.
Russell, Dan Abell, Medical Arts Bldg., San Antonio, Texas, ALR
Ryan, Maxwell D., 73 West 68th St., New York, N. Y., ALR
Sage, Russell A., 23 E. Ohio St., Indianapolis, Ind., ALR
Salerno, John Joseph, 214 E. 14th St., New York, N. Y., ALR
Saunders, Truman L., 103 E. 65th St., New York, N. Y., ALR
Schirack, Clarence John, 817 Cleveland Ave., N.W., Canton, Ohio, ALR

Schoofs, Orlando Peter, 324 E. Wisconsin Ave., Milwaukee, Wis., ALR
Seletz, Abraham, Medical Arts Bldg., Charleston, W. Va., ALR
Shafer, Albert Henry, 405 Cooper St., Camden, N. J., ALR
Shannon, James Bittick, 66 S. Fullerton Ave., Montclair, N. J., ALR
Shannon, Lardner Moore, 66 S. Fullerton Ave., Montclair, N. J., ALR
Sheppard, Ernest A. W., 927 Seventeenth St., Washington, D. C., Op.
Smith, Alma Ceshner, Marine Barracks, Quantico, Va., OALR
Smith, Eugene Churton, 811 Thomas Ave., Flint, Mich., ALR
Smith, Morley Thomas, 421 Huguenot St., New Rochelle, N. Y., ALR
Smith, William Hamilton, Arcade Bldg., Hagerstown, Md., ALR
Snyder, William Seth, Jr., McClure Bldg., Frankfort, Ky., ALR
Sperry, Frederick Noyes, 85 Whitney Ave., New Haven, Conn., ALR
Stevens, Joseph B., 1930 Wilshire Blvd., Los Angeles, Calif., ALR
Stovin, Joseph S., 160 W. 87th St., New York, N. Y., ALR
Strikol, Albert John, Medical Arts Bldg., Wilmington, Del., ALR
Symon, William Gardiner, The Clinic, Garrett, Ind., ALR
Teed, R. Wallace, First Nat'l Bank Bldg., Ann Arbor, Mich., ALR
Thorson, John A., Roshek Bldg., Dubuque, Iowa., Op.
Tickle, Thomas Gooch, 135 East 64th St., New York, N. Y., ALR
Town, Arno E., 140 E. 54th St., New York, N. Y., Op.
Tunstall, Cuthbart, 2nd and Market Sts., Charlottesville, Va., ALR
Usen, Louis, 276 New York Ave., Brooklyn, N. Y., ALR
Vaile, Ivan Royle, 455 Spadina Ave., Toronto, Ont., Can., ALR
Verhoeff, Frederick H., 243 Charles St., Boston, Mass., Op.
Vogel, Felix P. S., 1018 E. 163rd St., New York, N. Y., ALR
Voislawsky, Van Rensselaer, 33 E. 68th St., New York, N. Y., ALR
Wagner, Jesse LeVan, 152 N. Sixth St., Reading Pa., ALR
Weymuller, Ernest Alfred, 901 Lexington Ave., New York, N. Y., ALR
Whitney, Percy Theodore, 390 Main St., Worcester, Mass., Op.
Wilson, Hudson J., 156 E. State St., Ithaca, N. Y., ALR
Winston, Julius, 311 S. 20th St., Philadelphia, Pa., ALR
Wolpaw, Benjamin James, 2323 Prospect Ave., Cleveland, Ohio, Op.
Woodry, Norman Lee, 10 Witherell St., Detroit, Mich., ALR

Woodward, John F., Jr., 709 Park Ave., New York, N. Y., ALR
Youngs, Nelson A., Healy, Law, Woutat Clinic, Grand Forks, N. D., ALR
Zuckerman, Joshua, 108 East 91st St., New York, N. Y., Op.
Zwerling, Samuel, 789 St. Mark's Ave., Brooklyn, N .Y., ALR

* * *

Upon motion by Dr. Wherry, seconded by Dr. Fenton, the list of proposed members was accepted by the Academy.

Election of Officers

DR. WHERRY: Sir, on authority of the Council, we propose to nominate the following officers for the fiscal year of 1937:

President
LEE W. DEAN ..... St. Louis, Mo.

President-Elect
HARRY S. GRADLE ..... Chicago, Ill.

First Vice-President
BERNARD SAMUELS ..... New York, N. Y.

Second Vice-President
WILSON JOHNSTON ..... Portland, Ore.

Third Vice-President
F. L. RYERSON ..... Detroit, Mich.

Comptroller
S. H. LARGE ..... Cleveland, Ohio

Executive Secretary-Treasurer
WILLIAM P. WHERRY ..... Omaha, Nebr.

Secretary for Ophthalmology
WILLIAM L. BENEDICT ..... Rochester, Minn.

Secretary for Otolaryngology
JOHN L. MYERS ..... Kansas City, Mo.

Secretary for Instruction — Ophthalmology
HARRY S. GRADLE ..... Chicago, Ill.

Secretary for Instruction — Otolaryngology
DEAN M. LIERLE ..... Iowa City, Iowa

Secretary for Public Relations
RALPH A. FENTON ..... Portland, Ore.

Associate Secretaries
Ophthalmology — A. D. RUEDEMANN .... Cleveland, Ohio
Public Relations — ERLING HANSEN .. Minneapolis, Minn.

Member of Council
MARVIN F. JONES ..... New York, N. Y.

Member Board of Ophthalmology
JONAS S. FRIEDENWALD ..... Baltimore, Md.

Member Board of Otolaryngology
THOMAS E. CARMODY ..... Denver, Colo.
W. P. WHERRY ..... Omaha, Nebr.

American Committee on Optics and Visual Physiology
CHARLES A. BAHN ..... New Orleans, La.

Historian
JAMES A. BABBITT ..... Philadelphia, Pa.

American Association for the Advancement of Science
HARRY S. GRADLE ..... Chicago, Ill.

Honorary Chairman, 1937 Convention City
BURT R. SHURLY ..... Detroit, Mich.

General Chairman, 1937 Convention City
F. L. RYERSON ..... Detroit, Mich.

Upon motion by Dr. Hayden, seconded by Dr. Shurly, the nominations were accepted.

Drs. Lawrence Post, of St. Louis, and Dean Lierle, of Iowa City, escorted the President-Elect to the platform. The President asked the new President to make a few remarks.

DR. DEAN: When I think that since I attended the first meeting of this Society in 1898, in Chicago, that I have been more or less intimately associated with the Society since that time and have watched it grow from an infant to a real giant, when I think of those things, I deeply appreciate the honor which you have conferred on me.

The only thing I can say, and the only thing I can do is to help make the next session just as good as this convention.

The President-Elect was then asked to make a few remarks:

DR. GRADLE: Needless to say, I am supremely happy at this, which is the greatest honor that has ever come to me in my life, and probably the greatest honor I will receive. It is a strange feeling to be up on the platform at this Academy meeting facing the audience. So far, I have usually been in the back, behind the screens.

I am pleased at the kindness all of you have shown. I only hope I will be able to carry on and follow in the footsteps of my predecessors.

Selection of 1937 Convention City*

DR. WHERRY: Sir, the Council very seriously debated the meeting place for 1937. As is to be expected, a great many different cities desire the Academy. The Academy is most desirous of going to these different cities. However, if you will stop to realize a bit the necessities of the Academy, you know a cross-section of the entire membership must be seriously considered first in the selection of the city, and secondly in the selection of the proper hotel.

The Council, therefore, after seriously debating the question, selected Detroit, Michigan, for the 1937 Convention City.*

Upon motion by Dr. Post, seconded by Dr. Beck, the report was adopted to have Detroit the 1937 Convention City.

Closing

DR. BURCH: I wish to express my personal appreciation again of the fine cooperation it has been my experience to enjoy in working with the Council and with our Board of Secretaries and Officers, and also with the members of the New York Committee who have made this such a wonderful meeting. I extend to you my thanks for the privilege of presiding over the official gatherings and the Ophthalmic Section of this meeting.

The session adjourned at six forty-five o'clock.

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* Convention city for 1937 changed to Chicago, Illinois, instead of Detroit.

Directory

The American Academy
of Ophthalmology and Otolaryngology

Forty-First Annual Meeting
New York 1936

Alphabetical List of Members

* Attended 1936 meeting. Number in parentheses indicates year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

NOTICE: By action of the Council at the 1934 meeting:

- Fellows who have paid dues for thirty (30) years continuously will automatically become Life Members, with dues remitted.
- Fellows who have paid dues continuously for twenty-five (25) years will be termed Senior Members and their dues will be one-half the usual amount; at the present time, five dollars ($5.00) anually.
- All other Fellows (except Honorary) will be classified as Junior Members.

HONORARY FELLOWS

*Beck, Joseph C. ('01) .....185 N. Wabash Ave., Chicago, Ill., ALR
*Birkett, H. S.('06) ....1190 Mountain St., Montreal, Canada, ALR
Brunner, Hans('35) .Gumperdorfer St. 25, Vienna,VI,Austria, ALR
Dundas-Grant, Sir James.....148 Harley St., W. 1, London, Eng.
Elliot, Lt.-Col. R. H.('13)
54 Welbeck St., Cavendish Sq., W., London, Eng.
Elschnig, Prof. A.('12) . Ferdinandstrasse 10, Prague, Czechoslovakia
Foster, Hal('94) ..... Professional Bldg., Kansas City, Mo., ALR
Graves, Basil ('26)
11 Devonshire Place, Upper Wimpole St., W. 1, London, Eng.
Gray, Albert A.('11) ..... Glasgow, Scotland
de Grósz, Prof. Emil ('30) ..... Budapest, VIII, Maria, V. 39, Op.
*Jackson, Edward('02) ..... Republic Bldg., Denver, Colo., Op.
Johnson, Lindsay('05)
"Fairlawn" Esplanade, Durban, Natal, Africa, Op.
Kirkpatrick, Lt.-Col. Henry('24)
54 Welbeck St., W. 1, London, Eng.
Koller, Carl ('34) ..... 30 E. 58th St., New York, N. Y., Op.
Lauber, Prof. Dr. Hans('34) .... Aleja Roz 8, Warsaw, Poland, Op.
Lawford, J. B.('08) ... Sagamore, New Road, Ashtead, Surrey, Eng.
Lister, Sir William T.('23) .24 Devonshire Pl., W. 1, London, Eng.

NAGAR, PROF. F.('25) . . . . . Freiestrasse 20, Zurich, Switzerland
*PARSONS, SIR JOHN HERBERT('36)
54 Queen Anne St., Cavendish Sq. W., London, Eng., Op.
PATTERSON, MAJ. GEN. ROBERT U.('33)
U. S. Army, Office of Surgeon General, Washington, D. C., ALR
TILLEY, HERBERT('29) . . University College Hosp., London, Eng., Op.
VAN DER HOEVE, PROF. J.('21) . Rijnsburgerweg, 6 A, Leiden, Holland

LIFE MEMBERS

A

ADAMS, A. L.('06) . . . . . Professional Bldg., Jacksonville, Ill., OALR
*ADAMS, C. F.('06) . . . . . 34 W. State St., Trenton, N. J., OALR
ALLEN, J. H. . . . . 248 S. Michigan Ave., Pasadena, Calif., ALR
AMBERG, EMIL('06) . . . . . Maccabee Bldg., Detroit, Mich., ALR
ANDREWS, A. H.('08) . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR

B

BAKER, CHAS. H.('06) . Bay City Bank Bldg., Bay City, Mich., OALR
BALL, MICHAEL V.('04) . . . . . 316 Hazel St., Warren, Pa., OALR
BANE, WILLIAM C.('04) . . . . . Republic Bldg., Denver, Colo., Op.
BARNHILL, JOHN F.('03)
5369 Pine Tree Drive, Miami Beach, Fla., ALR
*BEATTIE, ROBERT('06) . . David Whitney Bldg., Detroit, Mich., OALR
*BEEBE, C. S.('07) . . 324 E. Wisconsin Ave., Milwaukee, Wis., Op.
BELLOWS, GEORGE E. ('96)
Professional Building, Kansas City, Mo., Op.
BENNETT, ARTHUR G.('05) . . 150 N. Pearl St., Buffalo, N. Y., Op.
BERNSTEIN, E. J.(1900) . . . . . 10 Peterboro, Detroit, Mich., OALR
BLAAUW, E. E.('05) . . . . . 190 Ashland Ave., Buffalo, N. Y., Op.
BLACK, MELVILLE('04) . 424 Metropolitan Bldg., Denver, Colo., Op.
BLACK, NELSON MILES('03) . . Huntington Bldg., Miami, Fla., Op.
BLACK, W. D.(1900) . . . . . Metropolitan Bldg., St. Louis, Mo., ALR
BLEDSOE, R. W.('07) . . . . 1005 Madison Ave., Covington, Ky., OALR
BRAWLEY, F. E.('07) . . . . . 30 N. Michigan Blvd., Chicago, Ill., Op.
*BROBST, C. H.('06) . . . . Central Nat'l Bank Bldg., Peoria, Ill., OALR
BROWN, EDWARD J. ('11)
3027 Pleasant Ave., Minneapolis, Minn., (Retired) OALR
BROWN, JOHN E.('01) . . . . . 370 East Town St., Columbus, O., OALR
BRUNER, WM. E.('05) . . . . . Guardian Bldg., Cleveland, O., Op.
BUCKMAN, E. U.('06) . 70 S. Franklin St., Wilkes-Barre, Pa., OALR
*BURCH, FRANK E.('07) . . . . . Hamm Bldg., St. Paul, Minn., Op.
BURKE, T. A.('06) . . . . . Rose Bldg., Cleveland, O., OALR

C

*CARMODY, T. E.('04) . . . . . Metropolitan Bldg., Denver, Colo., ALR
*CARTER, W. W. . . 6 Chestnut Ave., Bronxville, New York, N.Y., ALR
CHENERY, W. E. . . . 377 Commonwealth Ave., Boston, Mass., ALR
CLEVENGER, W. F.('05) . Hume-Mansur Bldg., Indianapolis, Ind., ALR
CULBERTSON, L. R.('98) . . . . Masonic Temple, Zanesville, O., OALR

D

DAVIS, J. LESLIE('06) . . . . . 135 S. 18th St., Philadelphia, Pa., ALR
DAY, EWING W.('08) . . 121 University Place, Pittsburgh, Pa., OALR
DEAN, F. W.('03) . . . . . 22 S. 6th St., Council Bluffs, Iowa, OALR

*Dean, L. W. ('98) . Washington U. Med. School, St. Louis, Mo., ALR
Dennis, F. L. ('07) . . . . . 2745 Barnson Pl., San Diego, Calif., ALR
Dixon, W. E. . . . . 1413 Spurgeon, Santa Ana, Calif., OALR
Doane, L. L. ('05) . . . . . 357 N. Main St., Butler, Pa., OALR
Dodd, Oscar ('03) . . . . . 636 Church St., Evanston, Ill., Op.
Donovan, John A. ('02) . . . 507 Phoenix Bldg., Butte, Mont., OALR

E

Easton, J. C. ('04) . . . First Nat'l Bank Bldg., Springfield, O., OALR
*Ellett, E. C. ('98) . . . . . Exchange Bldg., Memphis, Tenn., Op.
*Emerson, Linn ('06) . . . . . Metropolitan Bldg., Orange, N. J., Op.
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F

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HARLAN

GUNN, MILUS LYDELL ..... Smith Bldg., ALR

HOPKINSVILLE

GILMORE, MILTON A. ..... 901 Main St., Op.

LEXINGTON

ARMSTRONG, R. M. ..... First National Bank Bldg., OALR
DEWEESE, CLARENCE ..... 201 West Second St., OALR
MARKS, SAMUEL BLACKBURN ..... Security Trust Bldg., ALR
POOLE, WM. A. ..... Security Trust Bldg., ALR
YATES, EARL C. ..... 190 N. Upper St., ALR

LOUISVILLE

BASS, A. L. ..... Heyburn Bldg., OALR
DEAN, WALTER ..... 300 Francis Bldg., OALR
DULANEY, OCTAVIUS ..... 811 Brown Bldg., ALR
HALL, G. C. ..... 705 Brown Bldg., OALR
HEITGER, J. D. ..... Heyburn Bldg., OALR

LEGGETT, ALBERT E. .....614 Breslin Bldg., Op.
PFINGST, A. O. .....Heyburn Bldg., Op.
SHAFER, J. J. .....Starks Bldg., OALR
STERN, MILTON J. .....First Nat'l Bank Bldg., Op.
TOWNES, C. DWIGHT .....Heyburn Bldg., Op.
VAUGHAN, G. E. .....711 S. Third St., OALR
WOLFE, CLAUDE T. .....Heyburn Bldg., OALR

PADUCAH

PACE, ERRETT .....City National Bank Bldg., ALR
REYNOLDS, H. G. .....Citizens Savings Bank Bldg., OALR

RICHMOND

DUNN, MURISON .....OALR

LOUISIANA

LAFAYETTE

EDWARDS, SLOCOMB R. .....Commercial Bank Bldg., OALR

LAKE CHARLES

MOSS, OLIN W. .....Weber Bldg., ALR

NEW ORLEANS

ALEXANDER, LUCIAN W. .....1230 Maison Blanche Bldg., ALR
BAHN, CHAS. A. .....Pere Marquette Bldg., Op.
BLUM, HENRY N. .....American Bank Bldg., Op.
BREWSTER, H. F. .....Union Indemnity Bldg., ALR
BROWN, M. EARLE .....921 Canal St., Op.
BUFFINGTON, WILEY R. .....Hibernia Bank Bldg., Op.
CLARK, WM. B. .....American Bank Bldg., Op.
COX, CHAS. L. .....150 Baronne St., ALR
FUCHS, VALENTINE HENRY.....American Bank Bldg., ALR
GOOCH, JOHN B. .....Canal Bank Bldg., ALR
GRANBERRY, CARL .....Maison Blanche Bldg., ALR
HASPEL, M. DAVID .....Maison Blanche Bldg., OALR
HUME, JOHN R. .....Maison Blanche Bldg., ALR
LANDFRIED, C. J. .....607 Baronne Bldg., ALR
LEJEUNE, F. E. .....632 Maison Blanche Bldg., ALR
SMITH, VICTOR C. .....American Bank Bldg., Op.
TAQUINO, GEORGE J.....208 Audubon Bldg., ALR
WAGNER, W. A. .....200 Carondelet St., ALR
WALLS, ED. G. .....Maison Blanche Bldg., ALR
WEIL, A. I. .....3503 Prytania St., ALR
WOMACK, DAVID R. .....Maison Blanche Bldg., ALR

SHREVEPORT

LARUE, C. L. .....Highland Clinic, OALR
SCALES, J. L. .....Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W. ..... Masonic Temple, OALR

BANGOR

JOHNSON, JOHN L. ..... 33 Grove St., ALR

BATH

KERSHNER, W. E. ..... 117 Front St., ALR

LEWISTON

CHAFFERS, WM. H. ..... 190 Bates St., ALR

PORTLAND

BEACH, S. JUDD ..... 704 Congress St., Op.
CUMMINGS, GEORGE O. ..... 47 Deering St., ALR
FISHER, STANWOOD E. ..... 723 Congress St., ALR
GILBERT, FRANK Y. ..... 148 Park St., OALR
GORDON, CHAS. H. ..... 46 Deering St., ALR
HASKELL, ALFRED W. ..... 142 High St., Op.
HOLT, E. E., JR. ..... 723 Congress St., Op.
JOHNSON, HENRY P. ..... 32 Deering St., ALR
MCADAMS, WM. R. ..... 704 Congress St., Op.
O'SULLIVAN, T. J. ..... 10 Pine St., ALR
SPALDING, J. A. ..... 627 Congress St., Op.A

ROCKLAND

ELLINGWOOD, WM. A. ..... 420 Main St., ALR

WATERVILLE

HILL, FREDERICK T. ..... Professional Bldg., ALR
HILL, HOWARD F. ..... 179 Main St., Op.

MARYLAND

ANNAPOLIS

WELCH, ROBERT S. G. ..... 86 State Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR. ..... 300 N. Charles St., OALR
FRIEDENWALD, HARRY ..... 1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S. ..... 1212 Eutaw Pl., Op.
GOLDBACK, L. J. ..... 1012 N. Charles St., Op.
KEMLER, JOS. I. ..... 1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON ..... 104 W. Madison St., OALR
MACLEAN, ANGUS L. ..... 1201 N. Calvert St., Op.
PETERMAN, H. E. ..... 512 Cathedral St., OALR
TARUN, WILLIAM ..... 104 W. Madison St., Op.

CUMBERLAND

JONES, E. L. .....7 South George St., OALR
ROBINSON, H. T. .....Medical Bldg., OALR
SHARRETT, GEORGE O. .....119 Bedford St., OALR

HAGERSTOWN

SMITH, WM. H. .....Arcade Bldg., ALR

MASSACHUSETTS

BEVERLY

ODENEAL, THOMAS HELM .....21 Broadway, OALR

BOSTON

BRYANT, ALICE GERTRUDE .....405 Marlborough St., ALR
CAHILL, HARRY P. .....520 Commonwealth Ave., ALR
CASTEN, VIRGIL G. .....520 Commonwealth Ave., Op.
CHENERY, W. E. .....377 Commonwealth Ave., ALR
CLINE, SAMUEL .....353 Commonwealth Ave., ALR
COWAN, CLYDE R. .....Hotel Buckminster, ALR
DUNPHY, ED. B. .....101 Bay State Road, Op.
EVANS, MAURICE GEO. .....416 Marlborough St., ALR
FRAZEE, JOHN R. .....311 Beacon St., ALR
FRED, GUS B. .....475 Commonwealth Ave., ALR
FREEDMAN, L. M. .....475 Commonwealth Ave., ALR
GARLAND, FREDERICK E. .....483 Beacon St., LR
GOODALE, ROBT. L. .....258 Beacon St., ALR
GOODALL, EDWIN BAKER .....101 Bay State Road, Op.
GREENWOOD, ALLEN .....82 Commonwealth Ave., Op.
GUNDERSEN, TRYGVE .....5 Bay State Road, Op.
HALEY, WM. THOS. .....531 Beacon St., ALR
HARTSHORNE, ISAAC .....22 Commonwealth Ave., Op.
HOLMES, EDGAR M. .....520 Commonwealth Ave., ALR
HOOVER, WALTER BOYD .....605 Commonwealth Ave., ALR
HURLEY, E. D. .....419 Boylston St., Op.
INGLIS, HARRY J. .....43 Bay State Road, ALR
JOHNS, JUANITA PEARL .....313 Commonwealth Ave., Op.
KAZANJIAN, V. H. .....475 Commonwealth Ave., LR
KELLEY, VINCENT J. .....520 Commonwealth Ave., ALR
KLEINERT, MARGARET NOYES .....82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON .....520 Commonwealth Ave., ALR
LANCASTER, W. B. .....520 Commonwealth Ave., Op.
LEWIS, J. P. .....479 Commonwealth Ave., ALR
LOWELL, W. HOLBROOK .....82 Commonwealth Ave., Op.
LURIE, MOSES H. .....483 Beacon St., ALR
MELTZER, PHILIP E. .....20 Charlesgate West, ALR
MILLS, CHESTER R. .....520 Commonwealth Ave., ALR
MINITER, FRANCIS G. .....419 Boylston St., ALR
MOSHER, HARRIS P. .....828 Beacon St., ALR

MYSEL, PHILIP .....438 Beacon St., ALR
POIRIER, GEORGE H. .....60 Bay State Road, ALR
PORTER, CHAS. T. .....520 Commonwealth Ave., ALR
QUINCY, J. E. .....270 Commonwealth Ave., ALR
REGAN, JAS J. .....520 Commonwealth Ave., Op.
RICE, G. B. .....99 Bay State Road, ALR
RICHARDS, L. G. .....319 Longwood Ave., ALR
ROWLAND, W. D. .....84 Commonwealth Ave., Op.
SACHS, BENJAMIN .....483 Beacon St., Op.
SCHALL, LEROY A. .....270 Commonwealth Ave., ALR
SKIRBALL, J. J. .....353 Commonwealth Ave., Op.
TERRY, THEODORE L. .....128 Newbury St., Op.
THOMPSON, PETER H. .....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE .....416 Marlborough St., Op.
TOBEY, GEORGE L., JR. .....270 Commonwealth Ave., ALR
VERHOEFF, FREDERICK .....243 Charles St., Op.
WELLS, DAVID W. .....Hotel Westminster, Op.
WILKINS, SAMUEL HENRY .....270 Commonwealth Ave., Op.

CHICOPEE

GALLAGHER, JOHN H. C. .....105 Center St., ALR

CLINTON

MONAHAN, J. A. .....181 Chestnut St., OALR

FALL RIVER

BEST, LYNWOOD K. .....151 Rock St., Op.
RYDER, D. R. .....151 Rock St., ALR

FRAMINGHAM

TOMB, EVERETT H. .....46 Lexington St., ALR

GARDNER

BONE, HERMAN D. .....19 Pleasant St., OALR

GLOUCESTER

DEXTER, MILTON A. .....Cor. Pleasant and Prospect Sts., ALR
ODENEAL, THOMAS H. .....40 Middle St., OALR

GREENFIELD

MILLETT, FRANK A. .....58 Federal St., OALR

HAVERHILL

KAPP, HENRY .....50 Merrimack St., ALR

HOLYOKE

BURKE, JOHN R. .....207 Elm St., OALR

LAWRENCE

CONLON, F. A. .....301 Essex St., OALR

LOWELL

BERMAN, PHILIP G. .....174 Central St., Op.
LEAHEY, BRENDEN D. .....9 Central St., Op.
WIGGIN, WM. IRVING .....202 Central St., ALR

MALDEN

HAIRE, PAUL G. .....6 Pleasant St., Op.

LYNN

JONES, E. W. .....44 Atlanta St., Op. A
O'REILLY, W. F. .....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F. .....232 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D. .....187 Main St., ALR
DOW, FRANK E. .....4 West St., Op.

PALMER

MOORE, G. A. .....483 Main St., OALR

PITTSFIELD

THOMPSON, G. H. .....74 North St., OALR

SALEM

HENNESSEY, W. W. .....333 Essex St., OALR

SPRINGFIELD

BOSS, EUGENE G. .....1537 Main St., Op.
BYRNES, H. F. .....67 Chestnut St., OALR
CARLETON, RALPH .....10 Temple St., Op.
GAUDREAU, HONORE E. .....293 Bridge St., Op.
GOODELL, WILLIAM .....121 Chestnut St., ALR
IRWIN, VINCENT J., JR. .....1634 Main St., OALR
IRWIN, V. J., SR. .....1634 Main St. ,OALR
SEGAL, SAMUEL, JR. .....1562 Main St., ALR

TAUNTON

BAKER, HARRY B. .....59 Main St., OALR

WALTHAM

LEWIS, J. P. .....681 Main St., ALR

WORCESTER

BERRY, GORDON ..... 36 Pleasant St., ALR
CROISSANT, CHAS. A. ..... 390 Main St., ALR
CROSS, A. E. ..... 390 Main St., Op.
ESTABROOK, CHAS. T. ..... 10 Algonquin Road, OALR
GIBBY, HAROLD JAMES ..... 27 Elm St., ALR
GOODSPEED, HARRY B. ..... 36 Pleasant St., ALR
HOLZER, WM. F. ..... 36 Pleasant St., Op.
MESSIER, A. EUGENE ..... 390 Main St., OALR
MURPHY, WILLIAM E. ..... 390 Main St., ALR
RICE, JOHN E. ..... 390 Main St., OALR
WHITNEY, PERCY THEO. ..... 390 Main St., Op.

MICHIGAN

ALMA

HOWELL, D. M. ..... Polasky Bldg., ALR

ALPENA

NEWTON, WM. B. ..... 201 N. Second Ave., OALR

ANN ARBOR

BARR, ALBERT S. ..... First Nat'l Bank Bldg., Op.
FRALICK, F. BRUCE ..... First Nat'l Bank Bldg., Op.
FURSTENBERG, A. C. ..... First Nat'l Bank Bldg., ALR
MAXWELL, JAMES H. ..... First Nat'l Bank Bldg., ALR
TEED, R. WALLACE ..... First Nat'l Bank Bldg., ALR

BATTLE CREEK

DODGE, WARREN M., JR. ..... City Nat'l Bank Bldg., Op.
FRASER, R. H. ..... Security Nat'l Bank Bldg., ALR
HAUGHEY, WILFRED H. ..... Post Bldg., OALR
SLEIGHT, R. D. ..... Security Nat'l Tower, Op.
WENCKE, CARL G. ..... Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H. ..... Bay City Bank Bldg., OALR
SHERMAN, R. NEWTON ..... Phoenix Bldg., OALR

BENTON HARBOR

WESTERVELT, H. O. ..... 239 Pipestone St., ALR

BIRMINGHAM

FERRIS, RALPH G. ..... 55 W. Maple Ave., ALR

DETROIT

AMBERG, EMIL ..... 622 Maccabee Bldg., ALR
BARNETT, SAUL E. ..... 25 Parsons St., ALR
BEATTIE, ROBERT ..... David Whitney Bldg., OALR

BEGLE, HOWELL L. ..... David Whitney Bldg., Op.
BENTLEY, NEIL I. ..... David Whitney Bldg., OALR
BERNSTEIN, E. J. ..... 10 Peterboro, OALR
BROWN, AUDREY O. ..... 5057 Woodward, OALR
CAMPBELL, DON M. ..... David Whitney Bldg., OALR
CAMPBELL, DUNCAN A. ..... David Whitney Bldg., OALR
CAMPBELL, MALCOLM D. ..... David Whitney Bldg., OALR
CARTER, JOHN M. ..... David Whitney Bldg., ALR
CLIFFORD, T. P. ..... David Whitney Bldg., OALR
COHOE, D. A. ..... 13535 Woodward Ave., ALR
CROUSHORE, JAMES E. ..... 2501 W. Grand Blvd., ALR
DAVIES, WINDSOR S. ..... Eaton Tower, Op.
DEFNET, WM. A. ..... 28 Adams Ave. West, ALR
DOWLING, HARVEY E. ..... Eaton Tower, Op.
FOWLER, WM. ..... Maccabee Bldg., OALR
GEMEROY, JOSEPH C. ..... Henry Ford Hospital, ALR
GLOWACKI, BEN FRANCIS ..... Maccabee Bldg., OALR
GONNE, WM. S. ..... David Whitney Bldg., ALR
GOUX, RAYMOND SALOT ..... 545 David Whitney Bldg., OALR
GRANT, LEE E. ..... David Whitney Bldg., OALR
HALE, ARTHUR STANLEY ..... 1609 Eaton Tower, OALR
HAMMOND, ARTHUR ..... David Whitney Bldg., ALR
HARRELL, VOSS ..... Hofman Bldg., ALR
HEATH, PARKER ..... David Whitney Bldg., Op.
HOWELL, DONALD MACLEAN ..... David Whitney Bldg., ALR
HUGHES, RAY W. ..... 1501 David Whitney Bldg., OALR
KEATING, T. F. ..... 1042 Maccabee Bldg., OALR
KREUTZ, GEORGE C. ..... Henry Ford Hospital, ALR
MCCLELLAND, CARL C. ..... 1857 David Whitney Bldg., OALR
MERCER, R. E. ..... David Whitney Bldg., LR
MINER, S. G. ..... 2641 E. Jefferson Ave., ALR
MORTON, J. B. ..... 90 E. Warren Ave., OALR
MUNSON, F. T. ..... 2201 E. Jefferson Ave., ALR
O'HORA, B. A. ..... 3044 W. Grand Blvd., ALR
PARKER, WALTER R. ..... David Whitney Bldg., Op.
PEIRCE, HOWARD W. ..... David Whitney Bldg., ALR
PINO, RALPH H. ..... David Whitney Bldg., Op.
POOS, E. E. ..... Fisher Bldg., OALR
POTTER, WILLIS A. ..... 7450 Grand River Ave., ALR
RENAUD, G. L. ..... 510 Joseph Campan Ave., OALR
RYERSON, F. L. ..... Fine Arts Bldg., OALR
SANDERSON, H. H. ..... David Whitney Bldg., OALR
SCARNEY, HERMAN D. ..... 2501 W. Grand Blvd., Op.
SHURLY, BURT R. ..... 62 Adams Ave. W., ALR
SIMPSON, H. LEE ..... David Whitney Bldg., ALR
SUMMERS, WM. S. ..... 1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION ..... Fisher Bldg., ALR
WALKER, CHAS. C. ..... Maccabee Bldg., Op.
WENDEL, J. S. ..... 744 David Whitney Bldg., ALR
WHITNEY, E. L. ..... Henry Ford Hospital, ALR

WILLSON, W. W. .....2204 Eaton Tower, OALR
WOODRY, NORMAN LEE .....10 Witherell St., ALR
WOODWORTH, WILLIAM P. .....2994 E. Grand Blvd., OALR
WILKINSON, A. P. .....664 Fisher Bldg., ALR

FLINT

HUBBARD, WM. B. .....Dryden Bldg., ALR
SMITH, EUGENE C. .....811 Thomas Ave., ALR
STEVENSON, WM. W. .....Kresge Bldg., ALR

GRAND RAPIDS

DEAN, ALFRED .....308 Medical Arts Bldg., Op.
DEWAR, MURRAY M. .....Medical Arts Bldg., Op.
HEETDERKS, D. R. .....26 Sheldon S.E., ALR
LAIRD, ROBT. G. .....Metz Bldg., Op.
MCRAE, J. H. .....Medical Arts Bldg., Op.
MOLL, ARTHUR M. .....Powers Theatre Bldg., ALR
ROBERTSON, F. D. .....Ashton Bldg., OALR
SMITH, FERRIS .....Blodgett Medical Bldg., ALR
SNAPP, CARL F. .....Medical Arts Bldg., ALR

GROSSE POINTE VILLAGE

MERKEL, CHAS. C. .....279 Rivard Bldg., ALR

HOLLAND

HUIZINGA, J. G. .....Peoples State Bank Bldg., OALR

IONIA

SEDGWICK, O. W. .....OALR

IRON MOUNTAIN

BOYCE, G. H. .....Box 417, OALR

JACKSON

HARDIE, GEORGE C. .....290 W. Michigan Ave., ALR
MCGARVEY, WM. EDWARD. .....Jackson City Bank Bldg., ALR
WINTER, GEORGE E. .....Rogers Bldg., OALR

KALAMAZOO

FAST, RALPH B. .....American Nat'l Bank Bldg., ALR
FULKERSON, C. B. .....Room 403, 107 W. Michigan Ave., OALR
GRANT, FRED E. .....Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P. .....American Bank Bldg., OALR

LANSING

BEHEN, WM. C. .....221 N. Capitol Ave., ALR
BURDICK, AUSTIN F. .....330 N. Capitol Ave., OALR
ELLIS, C. WARD .....215 N. Walnut St., ALR
MCGILLICUDDY, OLIVER B. .....124 W. Allegan St., ALR
WETZEL, JOHN O. .....Olds Tower Bldg., Op.

MENOMINEE

JONES, WM. S. .....522 Sheridan Road, OALR

MT. CLEMONS

CROMAN, JOS. M., JR. .....117 Cass Ave., OALR

OWOSSO

POCHERT, ROLLAND C. .....Matthews Bldg., ALR

PETOSKEY

CONWAY, WM. S. .....314½ Howard St., ALR

PONTIAC

RIKER, AARON D. .....1012 Riker Bldg., Op.

PORT HURON

FRASER, ROBERT C. .....940 Military St., ALR

ST. JOSEPH

MCDERMOTT, JOHN J. .....1821 Niles Ave., OALR

SAGINAW

CADY, F. J. .....Peoples Bldg. and Loan Bldg., ALR
CORTOPASSI, ANDRE J. .....Second Nat'l Bank Bldg., ALR
MCKINNEY, A. R. .....704 Second Nat'l Bank Bldg., Op.
SLACK, WALTER K. .....308 Eddy Bldg., ALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK GARRETT ....Freeborn Co. Nat'l Bank Bldg., OALR

BRECKENRIDGE

RICE, CLARENCE PRENTICE .....OALR

CROOKSTON

MORLEY, GEORGE A. .....Crookston State Bank Bldg., Op.
OPPEGAARD, C. L. .....220 S. Broadway, ALR
PARSONS, J. G. .....Crookston Clinic, OALR

DULUTH

ARMSTRONG, E. LESLIE .....205 W. 2nd St., ALR
ATHENS, ALVIN G. .....Medical Arts Bldg., Op.
FELLOWS, M. FORDING .....Medical Arts Bldg., Op.
HILDING, ANDERSON C. .....Medical Arts Bldg., ALR
KNAPP, F. N. .....Medical Arts Bldg., OALR
SHASTID, T. H. .....202 W. Superior St., Op.
TILDERQUIST, D. L. .....Medical Arts Bldg., OALR
WINTER, JOHN A. .....205 W. Second St., OALR

FERGUS FALLS

COMBACHER, LEON C. ..... 101½ Lincoln Ave., W., Op.

HIBBING

MORSMAN, L. W. ..... Morsman Clinic, Op.
SINAMARK, ANDREW ..... 205 Power Bldg., OALR

MINNEAPOLIS

BOIES, LAWRENCE R. ..... Medical Arts Bldg., ALR
BORGESON, E. J. ..... Medical Arts Bldg., Op.
BROWN, EDWARD J. ..... 3027 Pleasant Ave., OALR
CAMP, WALTER E. ..... Medical Arts Bldg., OALR
CAMPBELL, ROBERT A. ..... 520 La Salle Bldg., OALR
CLARK, H. S. ..... Medical Arts Bldg., OALR
CURTAIN, JOHN F. ..... Medical Arts Bldg., ALR
DAHL, ELMER O. ..... Medical Arts Bldg., ALR
DORGE, RICHARD IRVING ..... 1111 Nicollet Ave., OALR
FINK, WALTER H. ..... Medical Arts Bldg., OALR
HANSEN, ERLING W. ..... Medical Arts Bldg., OALR
HOWARD, W. H. ..... Physicians and Surgeons Bldg., OALR
KOLLER, HERMANN M. ..... Medical Arts Bldg., Op.
KOLLER, LOUIS R. ..... Medical Arts Bldg., ALR
LITCHFIELD, J. T. ..... Medical Arts Bldg., OALR
LOOMIS, E. A. ..... Medical Arts Bldg., OALR
MACNIE, J. S. ..... Medical Arts Bldg., OALR
MATTHEWS, JUSTUS ..... Medical Arts Bldg., LR
NEWHART, HORACE ..... Medical Arts Bldg., ALR
OBERG, C. M. ..... Syndicate Bldg., OALR
PATTERSON, W. E. ..... Medical Arts Bldg., OALR
PHELPS, KENNETH A. ..... Medical Arts Bldg., OALR
PRATT, FRED J. ..... Physicians and Surgeons Bldg., OALR
PRATT, JOHN A. ..... Physicians and Surgeons Bldg., OALR
REYNOLDS, J. S. ..... Medical Arts Bldg., OALR
SCHWARTZ, VIRGIL JOSEPH ..... Medical Arts Bldg., ALR
SMITH, ARTHUR E. ..... Medical Arts Bldg., OALR
STROUT, E. S. ..... Medical Arts Bldg., OALR
STROUT, G. ELMER ..... 1009 Nicollet Ave., OALR
WATSON, JAMES A. ..... Medical Arts Bldg., OALR

NEW ULM

REINEKE, GEORGE F. ..... Fritsche Bldg., OALR

RED WING

BRUSEGARD, JAS. F. ..... 414 Main St., Op.

ROCHESTER

ANDERSON, CARL M. ..... Mayo Clinic, ALR
BENEDICT, WILLIAM L. ..... Mayo Clinic, Op.
FIGI, F. A. ..... Mayo Clinic, LR

HAVENS, FRED Z. ..... Mayo Clinic, ALR
HEMPSTEAD, BERT E. ..... Mayo Clinic, ALR
LILLIE, HAROLD I. ..... Mayo Clinic, ALR
NEW, GORDON B. ..... Mayo Clinic, L
PRANGEN, AVERY DE H. ..... Mayo Clinic, Op.
RUCKER, C. W. ..... 102 Second Ave., S.W., Op.
WAGENER, HENRY P. ..... Mayo Clinic, Op.

ST. CLOUD

WENNER, W. T. ..... St. Mary's Bldg., Op.

ST. PAUL

BERRISFORD, PAUL D. ..... Lowry Medical Arts Bldg., OALR
BINGER, H. E. ..... Lowry Medical Bldg., OALR
BOECKMANN, EGIL ..... Lowry Medical Arts Bldg., OALR
BROWN, JOHN C. ..... Medical Arts Bldg., OALR
BURCH, FRANK E. ..... Hamm Bldg., Op.
CONNOR, CHAS. E. .... Lowry Medical Arts Bldg., 4th St. Sec., ALR
FOGARTY, CHAS. W. ..... 372 St. Peter St., OALR
GRANT, H. W. ..... Lowry Bldg., Op.
HOCHFILZER, JOHN J. ..... Lowry Medical Arts Bldg., ALR
KENNEDY, WM. A. ..... 372 St. Peter St., Op.
LARSEN, CARL L. ..... 1027 Lowry Medical Bldg., OALR
LEAVENWORTH, R. O. ..... Lowry Medical Bldg., Op.
NELSON, ALLAN LOUIS ..... 830 Lowry Medical Arts Bldg., Op.
ROTHSCHILD, HAROLD J. ..... Lowry Bldg., OALR
SHELLMAN, J. L. ..... Lowry Medical Arts Bldg., OALR
WHEELER, MERRITT W. ..... 1027 Lowry Bldg., OALR
WOLD, K. C. ..... Lowry Medical Arts Bldg., OALR

WINONA

RISSER, EDWARD D. ..... 310 Choate Bldg., OALR
WALKER, GEORGE H. ..... 172 Main St., OALR

WORTHINGTON

STANLEY, C. R. ..... 508 Tenth St., ALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, D. CAMERON ..... Leyser Bldg., ALR

GULFPORT

MCWILLIAMS, CHAS. A. ..... G. & S. I. Bldg., ALR

JACKSON

ADKINS, GEORGE E. ..... Jackson Infirmary, ALR
HAGEMAN, VAN DYKE ..... Lamar Life Bldg., ALR
HARRIS, ROBIN ..... Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S. .....405 Franklin St., OALR

OXFORD

GUYTON, B. S. .....512 Van Buren Ave., OALR

VICKSBURG

EDWARDS, C. J. .....First National Bank Bldg., OALR
JONES, EDLEY H. .....1301 Washington St., OALR

MISSOURI

CARTHAGE

POST, WINFRED B. .....304 Grant St., OALR
POWERS, EVERETT .....305 S. Main St., OALR

COLUMBIA

SNEED, CARL M. .....Guitar Bldg., OALR

EXCELSIOR SPRINGS

SHOEMAKER, FERDINAND .....U. S. Veterans' Hospital, OALR

HANNIBAL

HORNBACK, GEORGE A. .....500 Broadway, ALR

KANSAS CITY

ALLEN, CHAS. E. .....712 Waldheim Bldg., OALR
ALTRINGER, A. N. .....Professional Bldg., ALR
BEAL, HOMER A. .....1002 Argyle Bldg., ALR
BEIL, J. W. .....Bryant Bldg., OALR
BELLOWS, GEORGE E. .....1500 Professional Bldg., Op.
BLACK, W. BYRON .....Professional Bldg., ALR
BLAKESLEY, T. S. .....Professional Bldg., OALR
CURDY, R. J. .....1102 Grand Ave., Op.
CURRAN, E. J. .....1805 Fed. Res. Bank Bldg., Op.
DAVIS, HARRY B. .....Bryant Bldg., OALR
DIXON, OTTO J. .....Professional Bldg., ALR
EUBANK, AMBROSE E. .....636 Argyle Bldg., OALR
FOSTER, HAL .....1426 Professional Bldg., ALR
GILLILAND, O. S. .....Professional Bldg., ALR
HEDRICK, HAROLD B. .....Professional Bldg., ALR
HOWARD, J. W. .....Professional Bldg., OALR
KEITH, W. E. .....Professional Bldg., OALR
KNIGHT, JOHN S. .....Professional Bldg., ALR
LEMOINE, A. N. .....Rialto Bldg., Op.
LORIE, ALVIN J. .....Commerce Bldg., ALR
LUX, PAUL .....2006 Bryant Bldg., ALR
MCALESTER, ANDREW W., JR. .....Bryant Bldg., Op.
MCALESTER, ANDREW W., 3RD .....Bryant Bldg., Op.

McCarty, Virgil W. ..... Professional Bldg., OALR
McKee, Joseph W. ..... Professional Bldg., Op.
Miller, Hugh ..... Argyle Bldg., OALR
Myers, John L. ..... 815 Shukert Bldg., OALR
Roberts, S. E. ..... Professional Bldg., ALR
Shumate, D. L. ..... 500 Argyle Bldg., OALR
Simpson, M. B. ..... Professional Bldg., ALR
Thomason, H. E. ..... Professional Bldg., ALR
Teall, Raymond E. ..... Professional Bldg., ALR
Yazel, Herman E. ..... 733 Rialto Bldg., OALR

POWERSITE

Coffelt, Theodore A. ..... OALR

ST. JOSEPH

Forgrave, L. Robert ..... 204 Logan Bldg., OALR
Minton, Wm. H. ..... Carby Bldg., OALR
Proud, Willard C. ..... Phys. and Surg. Bldg., OALR

ST. LOUIS

Alden, Arthur M. ..... Frisco Bldg., ALR
Alvis, Bennett Y. ..... 308 N. 6th St., Op.
Arbuckle, Millard F. ..... 539 N. Grand Ave., ALR
Bardenheier, F. G. A. ..... Frisco Bldg., ALR
Beisbarth, Carl C. ..... 3720 Washington Ave., Op.
Birsner, Louis J. ..... Missouri Bldg., ALR
Black, W. D. ..... Metropolitan Bldg., ALR
Boemer, L. C. ..... 3720 Washington Blvd., ALR
Brown, Clyde O. ..... 3720 Washington Blvd., ALR
Burns, S. S. ..... Missouri Theater Bldg., ALR
Calhoun, James Graham ..... Metropolitan Bldg., Op.
Charles, Jos. W. ..... Humboldt Bldg., Op.
Cone, Alfred J. ..... 4932 Maryland St., ALR
Costen, J. B. ..... Beaumont Medical Bldg., ALR
Dean, L. W. ..... Medical School, Washington Univ., ALR
Dobbs, Otto Ross ..... 2743 N. Grand Ave., ALR
Dyer, C. P. ..... 408 Humboldt Bldg., Op.
Eber, Carl Theodore ..... Carlton Bldg., Op.
Eimer, Charles Eugene ..... 316 Lister Bldg., ALR
Ferris, David P. ..... Missouri Theatre Bldg., ALR
Fineberg, Maxwell ..... Missouri Theatre Bldg., ALR
Freimuth, L. E. ..... University Club Bldg., ALR
Glick, Harry N. ..... 3115 S. Grand Blvd., ALR
Goldstein, M. A. ..... 4574 W. Papin St., ALR
Green, John ..... 3720 Washington Blvd., Op.
Greene, M. L. ..... Missouri Theater Bldg., Op.
Gundelach, C. A. ..... 825 University Club Bldg., ALR
Hansel, French Keller ..... 634 No. Grand Ave., ALR

HARDESTY, J. F. .....706 Missouri Theater Bldg., Op.
HARDY, W. F. .....Humboldt Bldg., Op
HILDRETH, H. ROMMEL .....Metropolitan Bldg., Op.
HOBART, CARL .....Metropolitan Bldg., Op
HOOSS, C. H. A. .....3115 S. Grand Blvd., Op.
HOURN, GEORGE E. .....513 University Club Bldg., ALR
HOWARD, HARVEY J. .....Park Plaza Hotel, Op.
JACOBS, MAX W. .....Missouri Theater Bldg., Op.
JAMES, WM. M. .....508 N. Grand Ave., Op.
JONES, VINCENT L. .....1242 Missouri Theater Bldg., OALR
KELLEY, ISAAC D., JR. .....3720 Washington Blvd., ALR
LAMB, HARVEY D. .....Metropolitan Bldg., ALR
LEAVY, C. A. .....Metropolitan Bldg., ALR
LUEDDE, W. H. .....Metropolitan Bldg., Op.
LYMAN, HARRY W. .....308 N. 6th St., ALR
MCMAHON, BERNARD JOHN .....Missouri Theater Bldg., ALR
MASON, ROY EARL .....Frisco Bldg., Op.
MILLER, H. ED. .....634 N. Grand Blvd., ALR
MILLIGAN, ROY H. .....3720 Washington Blvd., ALR
MYERS, E. LEE .....Wall Bldg., ALR
NEMOURS, PAUL ROLAND .....3142 Franklin Ave., ALR
PAYNE, R. J. .....University Club Bldg., ALR
PELZ, MORT D. .....Missouri Bldg., ALR
POOS, GROVER H. .....607 N. Grand Ave., OALR
POST, LAWRENCE T. .....Metropolitan Bldg., Op.
POST, M. HAYWARD .....Metropolitan Bldg., Op.
POTTER, ALLEN B. .....Pasteur Medical Bldg., ALR
PROETZ, A. W. .....Beaumont Bldg., ALR
REIM, HUGO .....417 Metropolitan Bldg., Op.
RYAN, L. M. .....Wall Bldg., ALR
SAUER, W. E. .....Beaumont Medical Bldg., ALR
SCHLUER, E. P. .....508 N. Grand Ave., ALR
SCHOLZ, R. P. .....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O. .....508 N. Grand Blvd., Op.
SENSENEY, EUGENE T. .....316 Lister Bldg., ALR
SHAHAN, W. E. .....Metropolitan Bldg., Op.
SHOEMAKER, W. A. .....1006 Carleton Bldg., Op.
SIMON, FREDERICK C. .....914 Arcade Bldg., ALR
SMIT, WM. M. .....5285 Washington Bvld., ALR
TOOKER, C. W. .....Carleton Bldg., Op.
WESTLAKE, SAMUEL B. .....Beaumont Med. Bldg., ALR
WIENER, MEYER .....900 Carleton Bldg., Op.
WOOD, V. VISSCHER .....Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E. .....Metropolitan Bldg., Op.

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SMITH, CARLIE SOUTER ..... Holland Bldg., OALR

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WEBSTER GROVES

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ANACONDA

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MORSE, A. W. ..... 507 Phoenix Bldg., OALR
POTTER, PETER ..... Motor Clinic Bldg., OALR

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MISSOULA

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BROOKS, EARL B. ..... 939 Stuart Bldg., OALR
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HOMPES, J. J. ..... Sharp Bldg., OALR
PETERSON, JOHN C. ..... Sharp Bldg., Op.
SANDERSON, DAVID D. ..... Stuart Bldg., OALR
THOMAS, JAMES W. ..... Sharp Bldg., OALR
ZEMER, S. G. ..... Stuart Bldg., OALR

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FAIER, SAMUEL Z. ..... Medical Arts Bldg., ALR
HANEY, WM. P. ..... 1500 Medical Arts Bldg., OALR
JUDD, DELBERT K. ..... Medical Arts Bldg., ALR
JUDD, J. H. ..... Medical Arts Bldg., Op.
KNODE, A. R. ..... 708 Medical Arts Bldg., ALR
KULLY, HERMAN E. ..... Medical Arts Bldg., ALR
POTTER, GEORGE B. ..... Medical Arts Bldg., OALR
POTTS, J. B. ..... Medical Arts Bldg., ALR
ROMONEK, PHILIP L. ..... Medical Arts Bldg., OALR
RUBENDALL, C. ..... Medical Arts Bldg., OALR
STOKES, H. B. ..... Medical Arts Bldg., ALR
STOKES, WM. H. ..... Medical Arts Bldg., Op.
SWAB, CHARLES M. ..... Medical Arts Bldg., Op.
UREN, C. T. ..... City National Bank Bldg., ALR
WHERRY, W. P. ..... 1500 Medical Arts Bldg., OALR

NEVADA

RENO

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UPHAM, HELEN F. .....305 Third Ave., OALR

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GORDON, BENJAMIN LEE .....1616 Pacific Ave., Op.
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SHAFER, ALBERT H. .....405 Cooper St., ALR
SHEMELEY, WILLIAM G., JR. .....7 Haddon Ave., OALR
SHIPMAN, JAMES S. .....542 Cooper St., Op.
SHOPE, PIERCE .....511 Cooper St., Op.

EAST ORANGE

BUVINGER, CHARLES W. .....50 Washington St., OALR

ELIZABETH

KAPP, CARL G. .....440 Westminster Ave., ALR

HACKENSACK

BERKE, RAYNOLD N. .....430 Union St., Op.
GREENFIELD, WILLIAM JOHN .....50 Anderson St., ALR

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PYLE, WALLACE .....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K. .....96 Third Ave., OALR

MONTCLAIR

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SHANNON, L. M. .....66 S. Fullerton Ave., ALR

NEWARK

ADELMAN, B. B. .....190 Clinton Ave., Op.
BARKHORN, CHAS. W. .....223 Roseville Ave., ALR

CARDWELL, EDGAR PARMELE .....47 Central Ave., ALR
CHATTIN, J. FRANKLIN .....671 Broad St., Op.
EAGLETON, WELLS P. .....15 Lombardy St., ALR
GOLDSTEIN, HENRY Z. .....31 Lincoln Park, ALR
HAHN, WM. H. .....15 Lombardy St., Op.
HUGHES, L. W. .....965 Broad St., OALR
HURFF, J. WALLACE .....86 Washington St., OALR
KEIM, WILLIAM F. .....25 Roseville Ave,. ALR
O'CONNOR, D. F. .....671 Broad St., OALR
ORTON, HENRY B. .....24 Commerce St., ALR
PEER, L. A. .....965 Broad St., ALR
QUINBY, W. O'G. .....14 James St., OALR
SHERMAN, A. RUSSELL .....671 Broad St., Op.
SHERMAN, ELBERT S. .....671 Broad St., Op.
WOOD, EARL LEROY .....160 Roseville Ave., ALR
ZEHNDER, A. CHARLES .....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN .....Metropolitan Bldg., Op.

PATERSON

ATWOOD, ED. A. .....360 Park Ave., ALR
PARK, M. BENJAMIN .....360 Park Ave., Op.

PERTH AMBOY

COHEN, NATHEN B. .....232 State St., ALR
MANN, J. J. .....255 State St., ALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON.....604 Park Ave., ALR
VAN HORN, A. F. .....514 Central Ave., ALR

RIDGEWOOD

DENISON, WARD C. .....20 E. 53rd St., ALR

SOUTH ORANGE

MILLER, JOSEPH A. .....364 Prospect St., ALR

SUMMIT

BURRITT, NORMAN W. .....30 Beechwood Road, ALR
SLY, JOHN L. .....382 Springfield Ave., ALR

TEANECK

LITTWIN, CHARLES .....962 Queen Anne Road, Op.

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ADAMS, C. F. .....34 W. State St., OALR

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SACCO, ANTHONY G. .....440 New York Ave., OALR

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SELINGER, SAMUEL .....413 Sixteenth St., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L. .....First Nat'l Bank Bldg., OALR

FORT BAYARD

GILL, ARTHUR E. .....U. S. Veterans' Hospital, OALR

ROSWELL

GRISWOLD, GEORGE WAYNE .....211 W. 3rd St., ALR

SANTA FE

BARTON, WM. C. .....104½ W. San Francisco St., Op.

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DOWLING, J. IVIMEY .....116 Washington Ave., OALR
HINMAN, E. E. .....146 Washington Ave., ALR
JUDGE, HARRY V. .....46 Willett St., Op.
SIMON, EMANUEL .....238 Lark St., ALR
TEBBUTT, H. K. .....240 State St., ALR

AUBURN

BULLARD, C. W. .....120 Genesee St., ALR

BATAVIA

WILSON, ROBERT G. .....433 E. Main St., ALR

BINGHAMTON

KINNE, SANFORD HALSEY .....73 Main St., ALR
RANDALL, HIRAM .....81 Main St., ALR
SMITH, WINDSOR R. .....67 Front St., ALR
WATSON, H. DE WITT .....151 Front St., OALR

BRONXVILLE

CARTER, WM. W. .....6 Chestnut Ave., ALR
MCKEOWN, HUGH S. .....Op.

BROOKLYN

AGAN, WM. B. ..... 510 Third St., Op.
ALBRIGHT, P. M., LT. COMDR. (M.C.) U.S.N.. U. S. Naval Hosp., OALR
ANDREW, JAS. H. ..... 163 Hancock St., Op.
BEHRENS, ALVIN ..... 9 Prospect Park West, Op.
BERGMANN, MILTON B. ..... 959 Bushwick Ave., Op.
BERNSTEIN, P. F. ..... 1265 President St., ALR
BLANKENFELD, NATHAN ..... 1506 Ocean Ave., ALR
BROOKS, MORRIS J. ..... 455 Ocean Ave., ALR
BRUNO, JOSEPH M. L. ..... 31 Jefferson St., Op.
BUONAGURO, MICHAEL J. ..... 589 Lorimer St., Op.
CUTINO, R. M. ..... 155 Henry St., Op.
DECOSTE, S. H. ..... 1179 Dean St., ALR
EBELING, WM. B. ..... 54 Greene Ave., Op.
EVANS, J. N. ..... 23 Schermerhorn St., Op.
FEASTER, HENRY J. ..... 461 77th St., ALR
FELDMAN, LOUIS A. ..... 259 New York Ave., Op.
FINE, ABRAHAM ..... 1505 Ocean Ave., Op.
GILBERT, JOS. GEO. ..... 50 Plaza St., ALR
GOLDEN, MATTHEW G. ..... 1 Nevins St., ALR
GOLDING, JOSEPH E. ..... 170 Hancock St., Op.
GREENFIELD, SAMUEL D. ..... 169 New York Ave., ALR
GREENFIELD, WM. J. ..... 777 West End Ave., ALR
GRIFFIN, EDWIN A. ..... 165 Hancock St., ALR
HARGITT, CHARLES A. ..... 17 Schermerhorn St., Op.
HOLZBERGER, JOHN C. ..... 7005 Freshpond St., ALR
HULL, ALLEN ..... 1847 Benson Ave., Op.
JAMESON, P. CHALMERS ..... 139 Montague St., Op.
KAPLAN, SAMUEL ..... 1 Nevins St., ALR
KEIL, PETER A. ..... 118 Pennsylvania Ave., ALR
KRIMSKY, EMANUEL ..... 745 Eastern Parkway, Op.
KRIMSKY, JOSEPH ..... 302 Empire Blvd., OALR
KRUSKAL, I. D. ..... 101 Lafayette Ave., Op.
LEIBOWITZ, PHILIP ..... 135 E. Parkway, ALR
LEVITT, JESSE M. ..... 734 Ocean Ave., Op.
LLOYD, RALPH I. ..... 14 Eighth Ave., Op.
LOMBARDO, MELCHIORE ..... 142 Joralemon St., Op.
LUPO, CARL W. ..... 7425 Ridge Blvd., ALR
MENGER, LOUIS CHARLES ..... 202 Weirfield St., ALR
MOEHLE, WALTER ..... 15 Schermerhorn St., Op.
MOORHEAD, ROBERT L. ..... 125 Remsen St., ALR
NOVA, JULES M. ..... 349 Adelphi St., Op.
O'BRIEN, G. R. ..... 307 Sterling Place, ALR
OHLY, JOHN H. ..... 22 Schermerhorn St., Op.
PLACE, E. CLIFFORD ..... 59 Livingston St., Op.
RABBINER, MAX ..... 358 New York Ave., ALR
RICHMAN, FRANCES ..... 134 St. Felix St., Op.
ROGERS, ROBERT M. ..... 680 St. Johns Pl., Op.
SCHILLINGER, RAPHAEL ..... 50 Plaza St., ALR

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SPARBER, HERMAN .....4701 Fifteenth Ave., Op.
STEINBUGLER, WM. F. C. .....515 Ocean Ave., Op.
THOMASHEFSKY, M. .....135 Eastern Parkway, ALR
USEN, LOUIS .....276 New York Ave., ALR
WECHSLER, A. H. .....340 New York Ave., ALR
WEETH, CHAS. R. .....37 Remsen St., ALR
DE YOANNA, ALFRED A. .....142 Joralemon St., ALR
ZWERLING, SAMUEL .....789 St. Mark's Ave., ALR

BUFFALO

ANDREWS, H. D. .....33 Allen St., Op.
BENNETT, ARTHUR G. .....150 N. Pearl St., Op.
BEYER, LOUIS J. .....449 Delaware Ave., ALR
BLAAUW, E. E. .....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE .....468 Delaware Ave., ALR
BROWN, C. M. .....510 Delaware Ave., ALR
BUFFUM, ED. S. .....40 W. North St., ALR
COTT, C. C. .....333 Linwood Ave., ALR
COWPER, H. W. .....543 Franklin St., Op.
EDSON, R. A. .....957 Delaware Ave., Op.
FAIRBAIRN, JOHN F. .....925 Delaware Ave., ALR
FLAGG, J. D. .....158 Anderson Place, Op. A
GLOSSER, HERBERT H. .....448 Franklin St., Op.
KOENIG, IVAN J. .....40 North St., Op.
LE WIN, THURBER .....112 Linwood Ave., Op.
LEWIS, F. PARK .....454 Franklin St., Op.
LUHR, A. F. .....40 W. North St., Op.
MCGREGOR, D'ARCY .....83 Bryant St., ALR
MCKEE, OTTO S. .....83 Bryant St., ALR
MARCH, CLARA A. .....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON .....131 Linwood Ave., Op.
RIWCHUN, MEYER H. .....367 Linwood Ave., Op.
ROSENBERG, JOSEPH .....333 Linwood Ave., ALR
TROTTER, HOMER A. .....40 North St., ALR
WEED, HARRY M. .....196 Linwood Ave., Op.
WURTZ, W. J. M. .....333 Linwood Ave., ALR

CORTLAND

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DUNKIRK

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ELMIRA

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FLUSHING

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GENEVA

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GLENS FALLS

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WILSON, HUDSON J. .....156 E. State St., ALR

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KEELER, LANSING H. .....89-04 148th St., OALR
MILLER, SAUL .....8805 150th St., Op.

JAMESTOWN

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WOODRUFF, WALTER STUART .....421 Huguenot St., ALR

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BASSEN, ED. J. .....654 Madison Ave., Op.
BEACH, B. S. .....140 W. 86th St., Op.
BELL, JULIUS W. .....307 E. 17th St., ALR
BENDER, M. S. .....30 E. 40th St., ALR
BERENS, CONRAD .....35 E. 70th St., Op.
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BERLINER, M. L. .....57 W. 57th St., Op.
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BOYES, T. L. .....654 Madison Ave., Op.
BRANIGAN, C. HUGH .....399 Park Ave., Op.
BRAUN, JACOB .....270 West End Ave., ALR
BRIGHTON, GEO. R. .....580 Park Ave., ALR
BROONES, OTTO .....302 W. 87th St., ALR
BUCKLEY, ROBT. .....50 E. 63rd St., ALR
BRUENING, PAUL H. .....133 E. 58th St., ALR
BRUCE, G. M. .....635 W. 165th St., Op.
BRUDER, JOSEPH .....57 W. 57th St., OALR
BULLWINKEL, H. GRIFFIN .....784 Park Ave., ALR
BUNNELL, CHESTER WM. .....375 Park Ave., ALR
BURMAN, HERMAN J. .....1235 Grand Concourse, ALR
CASTROVIEJO, RAMON .....635 W. 165th St., Op.
CINELLI, ALBERT A. .....1021 Park Ave., ALR
COHEN, MARTIN .....29 E. 64th St., Op.
COLBERT, ROBERT MARTIN .....110 W. 55th St., ALR
CONBOY, JOHN ED. .....2265 University Ave., Op.
CORYA, HAROLD W. .....30 E. 40th St., ALR
COTT, D. N. .....25 Central Ave., St. George, Staten Isl., ALR
CRACOVANER, ARTHUR J. .....103 E. 78th St., ALR
CUNNING, DANIEL S. .....145 E. 54th St., OALR
DAVIDSON, MORRIS .....80 Centre St., Dept. of Labor, Op.
DAVIS, A. EDWARD .....40 E. 61st St., Op.
DAVIS, G. E. .....1060 Fifth Ave., OALR
DEPPING, CHAS. WM. .....17 W. 54th St., ALR
DIXON, G. S. .....1150 Fifth Ave., R
DLUGASCH, LOUIS .....110 E. 36th St., ALR
DOHERTY, W. B. .....150 W. 55th St., Op.
DRUSS, JOS. GEO. .....1235 Park Ave., ALR
DUNNINGTON, J. H. .....30 W. 59th St., Op.
DWYER, JAS. G. .....300 Park Ave., ALR
ELLIS, Z. H. .....58 E. 65th St., Op.
FAULKNER, E. ROSS .....101 E. 58th St., ALR
FERNLUND, GEORGE BYRON ....8577 112th St., Richmond Hill, OALR

FELDMAN, WM. .....2021 Grand Concourse, ALR
FITZGERALD, FRED J. C. .....424 Madison Ave., Op.
FOSTER, HAROLD .....9205 — 35th Ave., Jackson Heights, ALR
FOWLER, ROBERT HENRY .....153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON .....12 E. 63rd St., ALR
FRANKEL, EDWARD, JR. .....217 E. 17th St., ALR
FREY, W. G., JR. .....121 E. 60th St., Op.
FRIEDMAN, BENJAMIN .....6 W. 77th St., Op.
FRIESNER, ISIDORE .....36 E. 73rd St., A
FURMAN, MARTIN A. .....142 W. 87th St., ALR
GARRICK, THOMAS J. .....40 E. 61st St., ALR
GATEWOOD, W. L. .....654 Madison Ave., ALR
GERSTLEY, MANFRED JOS. .....30 E. 40th St., ALR
GIVNER, ISADORE .....108 E. 66th St., Op.
GOLDMAN, A. MILTON .....121 E. 60th St., Op.
GOLDMAN, H. G. .....57 W. 58th St., ALR
GOLDSTEIN, ISADORE .....1160 Park Ave., Op.
GRAEBNER, HERBERT .....45 Park Ave., ALR
GRAEF, FRED W. .....850 Park Ave., ALR
GREENE, JAS. S. .....126 E. 30th St., ALR
GRESSER, EDWARD BELLAMY .....816 Fifth Ave., Op.
GRIFFITH, CHAS. M. .....140 E. 54th St., ALR
GRUSHLOW, ISRAEL .....271 Central Park, W., ALR
GUY, LOREN P. .....116 E. 62nd St., Op.
HALLETT, G. DEWAYNE .....274 W. 86th St., Op.
HARDY, LEGRAND H. .....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON .....104 E. 40th St., ALR
HARTSHORNE, ISAAC .....30 W. 59th St., Op.
HASKIN, W. H. .....300 Park Ave., ALR
HAYS, HAROLD M. .....133 E. 58th St., ALR
HETRICK, L. E. .....40 E. 61st St., ALR
HIRSCH, ALBERT .....125 E. 84th St., ALR
HIRSCH, CAESAR .....667 Madison Ave., ALR
HORN, WALTER L. .....1040 Park Ave., ALR
HOULAHAN, JAS. M. .....117 S. 2nd Ave., Op.
HURD, L. M. .....39 E. 50th St., ALR
HUNT, WESTLEY MARSHALL .....33 E. 68th St., ALR
HUTCHINSON, A. T. .....30 W. 59th St., ALR
HUVELLE, RENE H. .....17 E. 38th St., ALR
IMPERATORI, C. J. .....108 E. 38th St., ALR
INGERMAN, S. M. .....418 Central Park, W., OALR
JACOBS, S. M. .....1749 Grand Concourse, OALR
JARECKY, HERMAN .....120 W. 86th St., OALR
JEROME, WM. Z. .....436 Fort Washington Ave., ALR
JOHNSON, THOMAS HOLLAND .....30 W. 59th St., Op.
JONES, DAVID H. .....140 E. 54th St., LR
JONES, MARVIN F. .....121 E. 60th St., ALR
JOSEPHSON, EMANUEL M. .....108 E. 81st St., ALR
JUDD, H. B. .....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC .....140 E. 54th St., OALR

KEND, LEE .....405 Park Ave., ALR
KERNAN, JOHN D. .....103 E. 78th St., ALR
KEY, BEN WITT .....100 W. 59th St., Op.
KIRBY, D. B. .....780 Park Ave., Op.
KIRKBRIGHT, JAMES C. .....40 E. 61st St., ALR
KLEINFELD, LOUIS .....17 E. 84th St., ALR
KNAPP, ARNOLD .....10 E. 54th St., Op.
KNAPP, ARTHUR A. .....133 E. 58th St., Op.
KNIGHTON, W. S. .....40 E. 61st St., Op.
KOLLER, CARL .....30 E. 58th St., Op.
KOPETZKY, S. J. .....71 E. 80th St., ALR
KRUG, ERNEST F. .....988 Fifth Ave., Op.
LAMBERT, ROBERT K. .....1010 Fifth Ave., Op.
LAST, MURRAY A. .....1123 Park Ave., Op.
LA VIGNE, A. A. .....471 Park Ave., OALR
LEVINE, JOSEPH .....105 E. 63rd St., Op.
LIGGETT, HAROLD .....133 E. 58th St., ALR
LOEWE, WALTER R. .....1114 Madison Ave., Op.
LORE, JOHN M. .....325 West End Ave., ALR
LUBMAN, MAX .....118 E. 93rd St., ALR
MACDONALD, PHILIP G. .....115 E. 61st St., ALR
MACNIE, JOHN P. .....635 W. 165th St., Op.
MACPHERSON, DUNCAN .....140 E. 54th St., ALR
MCAULIFFE, GERVAIS W. .....110 W. 55th St., ALR
MCDANNALD, C. E. .....100 W. 59th St., Op.
MCKEOWN, HUGH S. .....30 W. 59th St., Op.
MALCOLM, PERRY E. D. .....410 Park Ave., ALR
MALINIAK, JACQUES W. .....1125 Park Ave., ALR
MARTIN, ASHBY G. .....540 Park Ave., ALR
MATUSOW, ABRAHAM P. .....1100 Grand Concourse, ALR
MAY, CHAS. H. .....70 E. 66th St., Op.
MAYBAUM, J. L. .....1070 Fark Ave., ALR
MEEK, R. E. .....729 Park Ave., Op.
MEIERHOF, E. L. (Retired) .....care Guaranty Trust Co., OALR
MILLER, JOSEPH WILLIAM .....311 E. 17th St., ALR
MINSKY, HENRY .....2 E. 95th St., Op.
MITTENDORF, A. D. .....399 Park Ave., Op.
MITTENDORF, W. KEITH .....115 E. 53rd St., Op.
MOGHTADER, MANSOOR .....40 E. 62nd St., ALR
MORRISON, WALLACE .....39 E. 50th St., ALR
MOSHINSKY, ALEXANDER L. .....237 E. 20th St., ALR
MOYLE, EUGENE H. .....1088 Park Ave., ALR
MYERSON, M. C. .....136 E. 64th St., ALR
NEAL, PHIL HAWKINS .....111 E. 75th St., ALR
NEER, EDMONDE DEWITTE .....115 E. 61st St., ALR
NISSELSON, MAX .....754 Beck St., ALR
NORTHINGTON, PAGE .....20 E. 53rd St., ALR
OAST, SAMUEL P., JR. .....30 E. 40th St., Op.
PALMER, ARTHUR .....667 Madison Ave., ALR
PAGE, JOHN R. .....127 E. 62nd St., ALR

PATON, R. T. ..... 927 Park Ave., Op.
PAYNE, B. F. ..... 35 E. 70th St., Op.
PERERA, CHAS. A. ..... 70 E. 66th St., Op.
PETERSON, R. A. ..... 150 Fifth Ave., OALR
PFEIFFER, RAYMOND L. ..... 635 W. 165th St., Op.
POSNER, ADOLPH ..... 667 Madison Ave., Op.
REESE, ALGERNON B. ..... 73 E. 71st St., Op.
RISCH, OTTO C. ..... 104 E. 40th St., ALR
ROSENBAUM, MORRIS ..... 61 W. 88th St., Op.
ROSENBLUTH, MICHAEL ..... 1018 E. 163rd St., ALR
ROSENWASSER, HARRY ..... 49 E. 96th St., ALR
ROSS, JOHN ..... 33 E. 68th St., ALR
RYAN, MAXWELL D. ..... 73 W. 68th St., ALR
SACKIN, DAVID ..... "St. Moritz," 50 Central Park South, ALR
SALERNO, JOHN J. ..... 214 E. 14th St., ALR
SAMUELS, BERNARD ..... 57 W. 57th St., Op.
SAUNDERS, TRUMAN L. ..... 103 E. 65th St., ALR
SCAL, JOSEPH COLEMAN ..... 27 W. 96th St., ALR
SCHECHTER, SOLOMON ..... 1749 Grand Concourse, ALR
SCHEER, HENRY M. ..... 522 West End Ave., ALR
SCHILLER, A. NOAH ..... 27 W. 72nd St., ALR
SCHLIVEK, KAUFMAN ..... 1016 Fifth Ave., Op.
SCHMIDT, OTTO V. M. ..... 111 E. 61st St., ALR
SCHOENBERG, MARK J. ..... 667 Madison Ave., Op.
SCHUGT, HENRY P. ..... 30 E. 40th St., ALR
SCHWARTZ, A. A. ..... 7 W. 71st St., ALR
SCHWARTZ, LEO, JR. ..... 133 E. 58th St., ALR
SETTEL, NATHAN ..... 10 W. 82nd St., ALR
SEWARD, J. A. ..... 115 E. 61st St., ALR
SITCHEVSKA, OLGA L. ..... 160 W. 87th St., Op.
SMITH, JAS. M. ..... 123 E. 53rd St., ALR
SOMBERG, J. S. ..... 876 Park Ave., Op.
STARK, JESSE B. ..... 45 Park Ave., Op.
STOVIN, JOSEPH S. ..... 160 W. 87th St., ALR
STRAATSMA, CLARENCE R. ..... 899 Park Ave., ALR
STURGES, LEIGH F. ..... 121 E. 60th St., ALR
TICKLE, THOMAS G. ..... 107 E. 63rd St., ALR
THYGESON, PHILLIPS ..... 630 W. 168th St., Op.
TOWN, ARNO E. ..... 140 E. 54th St., Op.
TUSAK, E. A. ..... 115 E. 90th St., Op.
VOGEL, FELIX P. S. ..... 1018 E. 163rd St., ALR
VOISLAWSKY, VAN RENSSELAER ..... 33 E. 68th St., ALR
WACHSBERGER, ALFRED ..... 521 Park Ave., ALR
WEEKS, WEBB WILLIAM ..... 20 E. 53rd St., Op.
WEINSTEIN, A. J. ..... 244 W. 74th St., ALR
WEXLER, DAVID ..... 179 E. 79th St., Op.
WEYMULLER, E. H. ..... 60 E. 75th St., ALR
WHEELER, J. M. ..... 635 W. 165th St., Op.
WHEELER, MAYNARD C. ..... 30 W. 59th St., Op.
WHELAN, WILLIAM F. ..... 2015 Walnut St., ALR

WHITE, JAMES W. .....15 Park Ave., Op.
WIENER, ALFRED .....550 Park Ave., OALR
WINCOR, HENRY G. .....1882 Grand Concourse, Op.
WOOD, ED. C. .....405 Park Ave., Op.
WOODMAN, DEGRAAF .....156 E. 37th St., ALR
WOODWARD, JOHN F., JR. .....709 Park Ave., ALR
ZUCKERMAN, JOSHUA .....108 E. 91st St., Op.

NIAGARA FALLS

PRICE, NORMAN W. .....445 Third St., OALR
ROOKER, A. M. .....United Bldg., ALR

NYACK

TOMS, S. W. S. .....120 S. Broadway, OALR

ONEIDA

CROCKETT, R. L. .....112 Farrier Ave., Op.

ONEONTA

HALL, EDWIN P. .....76 Chestnut St., ALR

OSSINING

NEWLANDS, WM. A. .....16 Maple Place, ALR

PLATTSBURG

SCHNEIDER, ANTON S. .....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A. .....103 Hooker Ave., OALR
VOORHEES, EARLE W. .....8 Garfield Place, ALR

ROCHESTER

AVERY, ELROY J. .....11 N. Goodman St., ALR
BARBER, F. .....75 S. Fitzhugh St., Op.
BARLOW, ROY A. .....Medical Arts Bldg., 277 Alexander St., ALR
BISHOP, VERNON L. .....277 Alexander St., ALR
CLARK, L. H. .....337 Monroe Ave., OALR
GIPNER, J. F. .....Medical Arts Bldg., Op.
JONES, L. W. .....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON .....Gas and Electric Bldg., Op.
LERNER, MACY LEVI .....332 Park Ave., Op.
MCDOWELL, N. D. .....275 Alexander St., OALR
MORRIS, A. G. .....Medical Arts Bldg., ALR
NASH, C. STEWART .....277 Alexander St., ALR
SHAPERO, I. M. .....289 Alexander St., OALR
SNELL, ALBERT C. .....53 S. Fitzhugh St., Op.

RYE

SOMBERG, J. S. .....127 Overdale Road, Greenhaven, Op.

SARANAC LAKE

BECK, ROBERT J. ..... 105 Main St., Op.

SYRACUSE

BRITTEN, G. S. ..... Medical Arts Bldg., ALR
BROWN, MORTIMER G. ..... 713 E. Genesee, ALR
BRUST, H. O. ..... 820 S. Crouse Ave., ALR
CAVE, LINUS S. ..... 221 Physicians Bldg., ALR
DICKINSON, WM. G. ..... State Tower, Op.
FOWLER, S. R. ..... University Block, ALR
GILLETTE, D. F. ..... State Tower Bldg., Op.
HALL, EDWIN P. ..... State Tower Bldg., ALR
HOOPLE, GORDON D. ..... Medical Arts Bldg., ALR
JOY, HAROLD HENRY ..... 504 State Tower Bldg., Op.
KAUFFMAN, A. B. ..... Chimes Bldg., ALR
KLINE, H. G. ..... State Tower Bldg., ALR
LEVY, HARRY H. ..... State Tower Bldg., Op.
LIVSHIN, NORMAN ..... University Bldg., ALR
MARLOW, SEARLE B. ..... 1004 State Tower Bldg., Op.
MOORE, ROY S. ..... State Tower Bldg., ALR
O'CONNOR, FREDERICK J. ..... Medical Arts Bldg., ALR
RUBENSTEIN, A. H. ..... Medical Arts Bldg., ALR
WEBSTER, F. R. ..... 720 State Tower Bldg., Op.

TARRYTOWN

NEWLANDS, W. ALEXANDER ..... 19 S. Broadway, ALR

TROY

SULZMAN, FRANK MALCOLM ..... 1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H. ..... 258 Genesee St., OALR
FARRELL, JAMES I. ..... 250 Genesee St., OALR
JONES, FRED G. ..... 258 Genesee St., ALR
VALONE, FRANK H. ..... 258 Genesee St., Op.

WATERTOWN

ATKINSON, JOHN H. ..... 168-70 Sterling St., ALR
ATKINSON, WALTER S. ..... 168 Sterling St., Op.
HENDERSON, L. E. ..... 168 Sterling St., Op.
MCASKILL, JAS. E. ..... Woolworth Bldg., ALR

WHITE PLAINS

MORIARTY, RICHARD W. ..... Medical Center, OALR
WOOD, ED. C. ..... 80 Maple Ave., Op.

WOODHAVEN

FROTHINGHAM, EDWARD ..... 8644 94th St., OALR

YONKERS

WOLCOTT, CHAS. C. ..... 27 Ludlow St., ALR

NORTH CAROLINA

ASHEVILLE

GREENE, JOSEPH BERRY .....Haywood Bldg., ALR
SWANN, CECIL C. .....706 Flatiron Bldg., ALR

CHARLOTTE

MATHESON, J. P. .....106 W. 7th St., ALR
PEELER, C. N. .....106 W. 7th St., OALR
SLOAN, HENRY L. .....106 W. 7th St., Op.
WHISNANT, A. M. .....517 Professional Bldg., OALR

DURHAM

ANDERSON, WM. B. .....McPherson Hospital, Op.
MCPHERSON, SAMUEL D. .....McPherson Hospital, ALR

GREENSBORO

REAVES, W. P. .....Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE .....Box 284, ALR

MOREHEAD CITY

JOHNSON, OTIS H. .....OALR

NEW BERN

DANIELS, R. L. .....Box 685, OALR

RALEIGH

HICKS, VONNIE MONROE .....Professional Bldg., Op.

WINSTON-SALEM

DAVIS, THOMAS W. .....210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

BUCKINGHAM, TRACY W. .....405 Broadway, ALR
CONSTANS, GEORGE MAURICE .....Clinic Bldg., Op.
ROSENBERGER, HENRY P. .....221 Fifth St., ALR

FARGO

FOSTER, GEO. C. .....807 Broadway, ALR
LARSON, G. ARTHUR .....A.O.U.W. Bldg., Op.
RINDLAUB, ELIZABETH P. .....de Lendrecie Bldg., OALR
RINDLAUB, JOHN H. .....de Lendrecie Bldg., OALR

GRAND FORKS

ALGER, L. JAS. .....First National Bank Bldg., ALR
YOUNGS, NELSON A. .....Healy, Law, Woutat Clinic, ALR

MINOT

LAMPERT, M. T. .....Northwest Clinic, ALR
MCCANNEL, A. D. .....15A Central Ave. W., OALR

OHIO

AKRON

ARBUCKLE, DAVID S. .....Akron Savings and Loan Bldg., OALR
BROWN, L. E. .....Second Nat'l Bank Bldg., OALR
BRUMBAUGH, JOHN D. .....Second Nat'l Bank Bldg., OALR
MOORE, T. K. .....Second Nat'l Bank Bldg., OALR
PARRY, THAYER L. .....Second Nat'l Bank Bldg., ALR
SEIDEL, U. D. .....Second Nat'l Bank Bldg., ALR

ALLIANCE

KING, GEORGE L., JR. .....537 E. Market St., Op.
KING, G. L., SR. .....537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. .....112 W. Columbus Ave., OALR
HARBERT, JOHN P. .....135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. .....309 Brant Bldg., ALR
SCHIRACK, CLARENCE J. .....817 Cleveland Ave., N.W., ALR
TOOT, JOSEPH F. .....First Nat'l Bank Bldg., Op.
WEAVER, HOMER V. .....228 Cleveland Ave., N.W., ALR

CINCINNATI

ABRAHAMSON, IRA R. .....925 Union Trust Bldg., Op.
ASBURY, MARY K. .....2466 Grandin Road, Op.
BEYER, A. G. .....Doctors Bldg., OALR
BROWN, ALBERT LOUIS .....Doctors Bldg., Op.
FISCHBACH, VICTOR W. .....Union Central Bldg., ALR
GOODYEAR, H. M. .....Doctors Bldg., ALR
HANS, C. L. .....Provident Bank Bldg., OALR
HARRIS, W. C. .....Groton Bldg., ALR
HENDRICKS, LOUIS J. .....210 W. 12th St., Op.
HOFMANN, ALBERT P. .....632 Vine St., ALR
HOFLING, CHARLES A. .....707 Race St., Op.
HOWARD, ROBERT E. .....Union Central Bldg., ALR
HYMAN, MORRIS .....Doctors Bldg., ALR
IGLAUER, SAMUEL .....707 Race St., ALR
JONES, C. C. .....Doctors Bldg., ALR

KING, ED. ..... 1002 Carew Tower, ALR
LOWENTHAL, GERSON ..... 19 Garfield Place, ALR
LYLE, DONALD J. ..... 909 Carew Tower, Op.
MARKS, JULIAN B. ..... 835 Carew Tower, ALR
MITHOEFER, W. ..... Doctors Bldg., ALR
PHINNEY, F. D. ..... 22 W. 7th St., OALR
RAY, VICTOR, JR. ..... Carew Tower, Op.
REID, H. W. ..... Union Central Bldg., Op.
SELTZ, SAMUEL ..... Provident Bank Bldg., ALR
SIEGEL, FRANCIS X. ..... 610 Union Central Life Bldg., OALR
STANBERY, HENRY ..... 566 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., ALR
STOLL, KARL LUDWIG ..... 19 West 7th St., OALR
SWING, FRANK U. ..... Doctors Bldg., ALR
TANGEMAN, H. F. ..... 606 Union Central Life Bldg., ALR
THOMPSON, E. H. ..... 19 W. 7th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR
VAIL, D. T. ..... Carew Tower, 441 Vine St., Op.
VAIL, H. H. ..... Carew Tower, 441 Vine St., ALR

CLEVELAND

ABBOTT, W. J. ..... Carnegie Medical Bldg., OALR
ALLEN, DONALD GRAY ..... 1021 Prospect Ave., Op.
BERNS, CHARLES ..... Osborn Bldg., ALR
BRUNER, ABRAM BRENEMAN ..... 1214 Guardian Bldg., OALR
BRUNER, WM. E. ..... Guardian Bldg., Op.
BUCHER, WALTER M. ..... 4187 Pearl Road, ALR
BURKE, T. A. ..... Rose Bldg., OALR
CHAMBERLAIN, WEBB PARKS ..... 7405 Detroit Ave., Op.
CUTLER, FRANKLIN E. ..... Hanna Bldg., ALR
DIRION, JOSEPHINE KOOB ..... Rose Bldg., Op.
DIXON, FRED WILLIS ..... Rose Bldg., ALR
ENGLER, CLARENCE WM. ..... 2323 Prospect Ave., ALR
FORSYTHE, S. T. ..... Rose Bldg., OALR
GARRETT, ERNEST W. ..... Hanna Bldg., ALR
JACOBY, MARK W. ..... Hanna Bldg., Op.
KENDALL, M. RAYMOND ..... 2474 S. Taylor Road, ALR
KINNEY, CHARLES ED. ..... 10515 Carnegie Ave., ALR
LARGE, S. H. ..... Hanna Bldg., ALR
LEMEL, SAMUEL L. ..... 1148 Euclid Ave., ALR
MCDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... Hanna Bldg., Op.
METZENBAUM, MYRON ..... Carnegie Medical Bldg., ALR
MITCHELL, HORACE E. ..... 14805 Detroit Ave., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Republic Bldg., Op.
MOORE, PAUL M., JR. ..... Cleveland Clinic, ALR
MOTTO, M. PAUL ..... Rose Bldg., Op.

MUSSUN, W. G. ..... Rose Bldg., ALR
NELSON, CHAS. F. ..... 606 Schofield Bldg., OALR
PITKIN, C. E. ..... Carnegie Medical Bldg., ALR
PUTNAM, F. I. ..... 1401 Prospect Ave., OALR
QUITTNER, S. S. ..... 10515 Carnegie Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... 10515 Carnegie Ave., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TUCKERMANN, W. C. ..... 733 Osborn Bldg., Op.
TUCKERMANN, W. H. ..... 733 Osborn Bldg., OALR
WAUGH, J. M. ..... Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. ..... 10515 Carnegie Ave., ALR
WOLPAW, BENJ. J. ..... 2323 Prospect Ave., Op.

COLUMBUS

BEATTY, H. G. ..... 150 E. Broad St., ALR
BROWN, JOHN E. ..... 370 E. Town St., OALR
CLARK, I. G. ..... 188 E. State St., OALR
DAVIES, MORGAN C. ..... 269 E. State St., Op.
DAVIS, WM. C. ..... 150 E. Broad St., Op.
FROST, ALBERT DALBEY ..... 150 E. Broad St., Op.
HARRIS, ED. WM. ..... 150 E. Broad St., ALR
HAUER, A. M. ..... 327 E. State St., ALR
HELFRICH, ED. D. ..... 269 E. State St., ALR
JOHNSON, DAVID M. ..... 327 E. State St., ALR
MEANS, R. G. ..... 40 S. Third St., ALR
PERRY, CLAUDE S. ..... 40 S. Third St., Op.
POSTLE, C. D. ..... 240 E. State St., ALR
PROUT, A. W. ..... 380 E. Town St., OALR
SANOR, DANIEL G., JR. ..... 206 E. State St., ALR
SILBERNAGEL, C. E. ..... 247 E. State St., OALR
STOUTENBOROUGH, WM. A. ..... 21 E. State St., OALR
THOMAS, FRANCIS W. ..... 327 E. State St., OALR
TIMBERMAN, ANDREW ..... Beggs Bldg., OALR

DAYTON

BEAVIS, JAMES O. ..... Fidelity Bldg., ALR
BINKLEY, ROY S. ..... Fidelity Bldg., OALR
BLACKBURN, W. J. ..... 663 Reibold Bldg., OALR
CULLER, ARTHUR M. ..... Fidelity Medical Bldg., Op.
DRISCOLL, FREDERICK JAS. ..... Reibold Bldg., ALR
DUTROW, H. V. ..... 1040 Fidelity Medical Bldg., OALR
FARMER, A. G. ..... Fidelity Medical Bldg., OALR
FOUTS, JOHN D. ..... Fidelity Medical Bldg., OALR

HARRIS, H. B. ..... Fidelity Medical Bldg., OALR
KILBOURNE, P. H. ..... Fidelity Medical Bldg., OALR
MILLETTE, J. W. ..... 117 S. Main St., OALR

FREMONT

RIDDELL, MALCOLM M. ..... 112½ Front St., ALR

LAKEWOOD

MERICA, F. W. ..... 14805 Detroit Ave., ALR
MILLER, G. LESLIE ..... 14805 Detroit Ave., Op.

LIMA

STUEBER, F. G. ..... 209 W. North St., OALR
STUEBER, PAUL J. ..... 209 W. North St., OALR

LORAIN

MONOSMITH, O. B. ..... O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M. ..... 14 N. Mulberry St., OALR
WISE, RALPH C. ..... News Bldg., OALR

MARIETTA

SAUER, W. W. ..... 231 Third St., OALR

MARION

BRICKLEY, DANIEL W. ..... Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E. ..... First Nat'l Bank Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H. ..... Castell Bldg., OALR

NEWARK

HATCH, C. B. ..... 3 W. Church St., OALR

PAINESVILLE

HEDLUND, GERALD O. ..... Nat'l Bank Bldg., ALR

SALEM

KING, LOWELL W. ..... 470 E. State St., ALR

SANDUSKY

KRAMER, JULIUS C. ..... 130 E. Market St., ALR

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EASTON, J. C. ..... First Nat'l Bank Bldg., OALR
HARTLEY, F. A. ..... 302 E. High St., OALR
HOGUE, D. W. ..... 34 W. High St., OALR

STEUBENVILLE

GOURLEY, G. F. ..... Sinclair Bldg., OALR

TIFFIN

PORTER, E. H. ..... 85 Madison St., OALR

TOLEDO

ALDERDYCE, WM. W. ..... 513 Madison Ave., OALR
BOICE, RALPH E. ..... 230 Michigan St., Op.
KING, CHAS. R. ..... Spitzer Bldg., OALR
LUKENS, CHARLES ..... 316 Michigan St., OALR
LUKENS, JOHN A. ..... 316 Michigan St., ALR
NIPPE, LEONARD ..... 316 Michigan St., OALR
SNYDER, W. H. ..... 2001 Colingwood Ave., OALR
STEINFELD, A. L. ..... 125 Fifteenth St., OALR
WINGER, IRA B. ..... 709 Madison Ave., OALR

VAN WERT

EDWARDS, STEPHEN A. ..... Home Guard Bldg., ALR

WARREN

STIMSON, GEORGE W. ..... Mahoning Bldg., OALR

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MADDEN, REED ..... Allen Bldg., OALR
SHIELDS, LAWRENCE ..... Allen Bldg., ALR

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EVANS, WILLIAM H. ..... Dollar Bank Bldg., ALR
GOLDCAMP, ED. C. ..... Dollar Bank Bldg., ALR
HARTZELL, SOL. M. ..... Dollar Bank Bldg., OALR
KEYS, JOHN E. L. ..... 613 Home Savings and Loan Bldg., Op.
WALKER, ORVILLE J. ..... Home Savings and Loan Bldg., ALR

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ADA

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ENID

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FORT SILL

CULPEPPER, WM. S. ..... Station Hospital, ALR

GUTHRIE

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FULLENWIDER, C. M. .....Barnes Bldg., OALR

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GUTHRIE, A. L. .....1200 N. Walker St., OALR
MACDONALD, J. C. .....301 N. W. 12th St., ALR
MCHENRY, D. D. .....Medical Arts Bldg., OALR
MCHENRY, L. CHESTER .....Medical Arts Bldg., ALR

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BRASWELL, JAMES C. .....Medical Arts Bldg., OALR
COOK, W. ALBERT .....Medical Arts Bldg., OALR
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HUBER, WALTER A. .....Medical Arts Bldg., Op.
ROTH, ALBERT W. .....Medical Arts Bldg., OALR
SMITH, RURIC N. .....Medical Arts Bldg., ALR

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BAKER

GORDON, GLENN G. .....Alexander Bldg., ALR

EUGENE

NORTON, ARTHUR H. .....1162 Williamette, Op.

LA GRANDE

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RALSTON, FRANK L. .....Sacajawea Annex, ALR

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BROWNING, ANDREW J. .....Medical Arts Bldg., Op.
CARRUTH, HOWARD ERNEST .....Medical Arts Bldg., OALR
DAVIS, RALPH F. .....Medical Arts Bldg., OALR
FENTON, RALPH A. .....806 Medical Arts Bldg., OALR
HENTON, G. E. .....720 S. W. Washington St., OALR
JOHNSTON, WILSON .....1927 N. W. Lovejoy St., OALR
KARKEET, ROBERT B. .....833 S. W. 11th Ave., OALR
KIEHLE, F. A. .....1020 S. W. Taylor St., OALR
KISTNER, F. B. .....600 Mayer Bldg., OALR
LUCAS, HAROLD R. .....Medical-Dental Bldg., OALR

LUPTON, IRVING MARTIN .....806 Medical Arts Bldg., OALR
NEELY, PAUL T. .....Medical Arts Bldg., ALR
PETHERAM, C. C. .....Medical Arts Bldg., ALR
TAYLOR, EDGAR M. .....Medical Arts Bldg., Op.
WEEKS, JOHN E. .....360 Edgecliff Road (Retired), Op.

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BAUM, WM. W. .....Guardian Bldg., ALR
CLEMENT, L. O. .....Guardian Bldg., OALR

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UHLER, S. MANN .....104 N. Eighth St., ALR

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BUZZARD, JOSIAH F. .....1110 Thirteenth Ave., Op.
GLOVER, S. P. .....1118 Twelfth Ave., OALR

ARDMORE

BOSTWICK, D. S. .....106 Linwood Ave., ALR
FURLONG, THOS. F., JR. .....Times Medical Bldg., ALR
MCANDREWS, LEO F. .....Times Medical Bldg., Op.

BEAVER

CRUMRINE, NORMAN ROSS .....595 Third St., OALR
SCROGGS, J. J. .....218 E. Third St., Op. A

BETHLEHEM

CHASE, W. D. .....230 E. Broad St., ALR

BUTLER

DOANE, L. L. .....357 N. Main St., OALR
HAZLETT, L. R. .....Savings and Trust Bldg., OALR
HUNT, GUY ANDERSON.....Butler Co. Nat'l Bank Bldg., Op.

CARLISLE

NEBINGER, REID .....156 W. Pomfret St., ALR

CHESTER

CROSS, G. H. .....525 Welsh St., Op.
ORR, ADRIAN V. B. .....525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H. .....308 First Nat'l Bank Bldg., OALR
MCLELLAN, THOMAS G. .....First Nat'l Bank Bldg., Op.

DANVILLE

DAVISON, FRANCIS WELLS .....Geisinger Hospital, ALR
MARSHALL, DON .....Geisinger Memorial Hospital, Op.

DU BOIS

BEALS, C. WEARNE ..... 41 N. Brady St., ALR

EASTON

BLOOM, C. HUGH ..... 65 N. Third St., ALR

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FERMAN, JOHN W. ..... OALR

ERIE

DELANEY, JAS. H. ..... 138 W. 9th St., Op.
DENNIS, EDWARD ..... 309 W. 10th St., OALR
FUST, JOHN H. E. ..... 231 W. 7th St., OALR
JACKSON, J. D. ..... 232 W. 8th St., OALR
RUSSELL, J. A. MERLE ..... 813 Sassafras St., OALR
SCHLINDWEIN, G. WM. ..... 138 W. 9th St., OALR
SHREVE, OWEN M. ..... 162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B. ..... West Park and Liberty St., OALR

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FAIRING, JOHN W. ..... 118 N. Main St., OALR
McKEE, CLAUD W. ..... Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L. ..... 618 N. 3rd St., OALR
GRACEY, GEORGE F. ..... 209 State St., OALR

HAZLETON

REICHE, O. C. ..... Markle Bank Bldg., Op.

HUNTINGDON

HUNT, WILLIAM T., JR. ..... 514 Penn St., ALR

JOHNSTOWN

BARKER, O. G. A. ..... 804 Johnstown Bank and Trust Bldg., Op.
GRIFFITH, HAROLD M. ..804 Johnstown Bank and Trust Bldg., Op.
HARRIS, C. M. ..... 604 Johnstown Bank and Trust Bldg., OALR

LANCASTER

LEFEVRE, WILLIS H. ..... 234 N. Duke St., ALR

LANSDOWNE

BUSLER, HOWARD S. .... N.E. cor. Baltimore and Owen Aves., ALR

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ALLIS, PAUL M. ..... 6 N. Brown St., ALR

MIDDLETOWN

GEORGE, HENRY W. .....19 N. Union St., Op.

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STEIM, J. M. .....First Nat'l Bank Bldg., Op.

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PRICE, JOHN BEADLE .....824 De Kalb St., ALR

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ADLER, FRANCIS HEED .....313 S. 17th St., Op.
ALEXANDER, GEORGE J. .....1930 Chestnut St., ALR
ANNON, W. T. .....235 S. 15th St., ALR
APPLEMAN, L. F. .....308 S. 16th St., Op.
BABBITT, JAMES A. .....1912 Spruce St., ALR
BAER, BENJAMIN F., JR. .....1927 Spruce St., Op.
BALENTINE, P. L. .....1524 Chestnut St., Op.
BALL, SIMON .....311 S. 20th St., ALR
BARLOW, AARON .....1307 Spruce St., Op.
BATSON, OSCAR V. ....Graduate School of Med., Univ. of Pa., ALR
BLAIR, MORTIMER W. .....N.E. Cor. 20th and Chestnut Sts., Op.
BOEHRINGER, H. WINFIELD.....700 Central Medical Bldg., OALR
BRICKER, SACKS .....1101 W. Wyoming Ave., ALR
BRIGLIA, FRANK J. .....703 Wharton St., ALR
BROWN, SAMUEL HORTON .....1930 Chestnut St., Op.
BRUMM, SETH A. .....Medical Arts Bldg., OALR
BURNS, L. J. .....1930 Chestnut St., ALR
BUTT, MIRIAM M. .....2045 Chestnut St., Op.
CARPENTER, C. .....1930 Chestnut St., Op.
CLERF, LOUIS HENRY .....1530 Locust St., ALR
COATES, GEORGE M. .....1721 Pine St., ALR
COHEN, HERMAN B. .....255 S. 17th St., ALR
COHEN, LOUIS B. .....1521 Arrott St., Op.
COHEN, SAMUEL .....2009 Pine St., ALR
COWAN, ALFRED .....1930 Chestnut St., Op.
CREIGHTON, WM. J. .....Physicians' Bldg., Op.
DAVIS, J. LESLIE .....135 S. 18th St., ALR
DAVIS, OSCAR .....U. S. Naval Hospital, OALR
DAVIS, T. CARROLL .....3128 N. Broad St., ALR
DAVIS, WARREN B. .....135 S. 18th St., ALR
DEICHLER, L. WALLER .....1930 Chestnut St., OALR
DINTENFASS, HENRY .....1305 Spruce St., ALR
DUNN, LOUIS S. .....255 S. 17th St., ALR
EMBERY, FRANK .....4801 Penn St. (Frankford) ALR
ERSNER, MATTHEW S. .....1915 Spruce St., ALR
EVES, C. C. .....1910 Spruce St., ALR
FEWELL, A. G. .....1924 Pine St., Op.
FRY, W. E. .....1819 Chestnut St., Op.

GIBSON, GLEN G. .....3701 N. Broad St., Op.
GODDARD, H. M. .....1531 Spruce St., ALR
GORDON, WM. .....1721 Pine St., ALR
GOUTERMAN, JOSEPH I. .....1930 Chestnut St., Op.
GOWEN, FRANCIS V. .....904 E. Chelton Ave., ALR
GRIMES, CHAS. H. .....3546 N. Broad St., ALR
GRISCOM, J. MILTON .....255 S. 17th St., Op.
HAWMAN, ERLE G. .....4051 N. Broad St., Op.
HEED, C. R. .....1205 Spruce St., Op.
HEWSON, WM. .....6013 Greene St., Germantown, ALR
HIRST, OTTO C. .....255 S. 17th St., ALR
HOUSER, KARL M. .....2010 Spruce St., ALR
HUNTER, R. J. .....2011 Chestnut St., ALR
HUSIK, D. N. .....1930 Chestnut St., ALR
IVY, ROBERT H. .....1930 Chestnut St., ALR
JACKSON, CHEVALIER .....3432 N. Broad St., L
JACKSON, CHEVALIER L. .....3432 N. Broad St., L
KAUFMAN, A. SPENCER .....1923 Spruce St., ALR
KELLY, GEORGE F. J. ..Suite 700, N.E. Cor. 20th and Chestnut, Op.
KRAUSS, FREDERICK .....1930 Chestnut St., OALR
LAESSLE, HENRY A. .....5900 Market St., ALR
LAWRENCE, GRANVILLE A. .....Medical Arts Bldg., Op.
LEWIS, FIELDING O. .....259 S. 17th St., ALR
LILLIE, WALTER I. .....3701 N. Broad St., Op.
LOFTUS, JOHN EDWARD .....1930 Chestnut St., ALR
LUKENS, ROBERT McD. .....1923 Spruce St., ALR
LUONGO, R. A. .....1713 Pine St., ALR
MACKENZIE, GEORGE W. .....269 S. 19th St., OALR
MANN, BENJAMIN H.....N.E. Corner 20th and Chestnut Sts., Op.
MILLER, M. VALENTINE....114 W. Phil-Ellena St., Mt. Airy, ALR
MITCHELL, E. K. .....710 W. Lehigh Ave., ALR
MOORE, WILLIAM FREDERICK .....255 S. 16th St., ALR
MYERS, DAVID .....2109 S. 16th St. and 1915 Spruce St., ALR
O'BRIEN, THOMAS A. .....1727 W. Girard Ave., Op.
OLSHO, S. L. .....235 S. 15th St., OALR
OWSLEY, JOHN Q. .....U. S. Naval Hospital, ALR
PERSKY, ABRAM H. .....1723 Pine St., ALR
PETER, L. C. .....1930 Chestnut St., Op.
PONTIUS, P. J. .....1831 Chestnut St., Op.
REESE, WARREN S. .....230 S. 21st St., Op.
RIDPATH, ROBERT F.....N.E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C. .....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON .....1924 Pine St., Op.
SARTAIN, PAUL J. .....2006 Walnut St., Op.
SCARLETT, HUNTER W. .....230 S. 21st St., Op.
SCHWEINITZ, GEORGE E. DE .....1705 Walnut St., Op.
SENER, WALTER J. .....Physicians Bldg., Op.
SHANNON, C. E. G. .....1930 Chestnut St., Op.
SHUSTER, BENJAMIN HARRISON .....1824 Pine St., ALR
SKILLERN, S. R., JR. .....1734 Pine St., ALR

SLOANE, HENRY O. .....1717 Pine St., Op.
SPAETH, EDMUND B. .....1930 Chestnut St., Op.
SPENCER, W. H. .....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M. .....1831 Chestnut St., Op.
STOUT, PHILIP S. .....269 S. 19th St., ALR
TASSMAN, ISAAC S. .....Medical Arts Bldg., Op.
TOWSON, CHAS. E.
Germantown Prof. Bldg., Greene and Coulter Sts., ALR
TUCKER, GABRIEL .....326 S. 19th St., B
WAGERS, ARTHUR J. .....1429 Spruce St., ALR
WEBER, LT. COMDR. HENRY C. (M.C.) ...U. S. Naval Hospital, OALR
WEINSTEIN, M. A. .....1817 Spruce St., ALR
WHELAN, G. L. .....2100 Walnut St., ALR
WHELAN, WILLIAM F. .....1925 Spruce St., ALR
WILLIAMS, HORACE J. .....5908 Greene St., Germantown, ALR
WINSTON, JULIUS .....311 S. 20th St., ALR
ZENTMAYER, WILLIAM .....1930 Chestnut St., Op.

PITTSBURGH

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BILLINGS, ROBERT J. .....500 Penn Ave., Op.
BLAIR, W. W. .....121 University Pl., Op.
CARSON, W. E. .....Jenkins Arcade Bldg., Op.
CONWAY, JOHN M. .....5151 Jenkins Arcade, ALR
COOPER, ROY C. .....510 S. Aiken Ave., ALR
CURRY, GLENDON E. .....Westinghouse Bldg., Op.
DAY, KENNETH M. .....121 University Pl., ALR
DAY, EWING W. .....121 University Pl., OALR
FISCHER, N. ARTHUR .....623 Union Trust Bldg., ALR
GROSS, JULIUS E. .....3401 Fifth Ave., ALR
GUERINOT, A. J. .....604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A. .....Highland Bldg., ALR
HAMPSEY, A. R. .....806 May Bldg., ALR
JENNINGS, CHAS. W. .....121 University Place, Op.
KREBS, ADOLPH .....Jenkins Arcade Bldg., Op.
LINN, J. G. .....Jenkins Arcade Bldg., Op.
MCCOLLOUGH, THOMAS B. .....121 University Pl., ALR
MCCREADY, J. HOMER .....121 University Pl., ALR
MACLACHLIN, A. A. .....May Bldg., ALR
MARKEL, J. CLYDE .....Westinghouse Bldg., Op.
MARSHALL, WATSON .....121 University Pl., ALR
MEANOR, WM. C. .....1715 Clark Bldg., Op.
MOON, SEYMOUR B. .....200 Ninth St., Op.
MOYER, JOHN S. .....303 Coltart Ave., ALR
PATTERSON, ELLEN J. .....121 University Pl., ALR
PLUMER, JOHN S. .....121 University Pl., Op.
REED, CHAS. L. .....Jenkins Bldg., Op.
RIMER, FRANK H. .....Jenkins Arcade, ALR
RUBIN, NATHAN S. .....Blount Bldg., Op.

SEEGMAN, SIMON .....Medical Bldg., ALR
SHUMAN, G. H. .....Park Bldg., Op.
SIMPSON, JOHN REID .....Medical Arts Bldg., ALR
STIEREN, EDWARD .....626 Union Trust Bldg., Op.
TAYLOR, M. C. .....May Bldg., Op.
THORPE, H. E. .....8075 Jenkins Arcade Bldg., Op.
TODD, GROVER CLEVELAND .....Jenkins Arcade Bldg., ALR
TURNER, H. H. .....Clark Bldg., Op.
WEISSER, E. A. .....806 May Bldg., Op.
WIBLE, LEROY E. .....Empire Bldg., ALR
YOST, WALTER MILTON .....Westinghouse Bldg., Op.

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CRESS, WILLIAM J. .....318 W. Market St., ALR

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HIGH, ISAAC B. .....326 N. 5th St., ALR
LANDIS, JAMES E. .....517 Walnut St., ALR
RHODE, SOLON L. .....238 N. 6th St., OALR
WAGNER, JESSE LEVAN .....152 N. 6th St., ALR

RIDGWAY

MCALLISTER, J. C. .....10 S. Mill Ave., AL

ROCHESTER

YOST, WALTER M. .....216 Brighton Ave., Op.

SAYRE

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SCRANTON

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CURTIN, EUGENE A. .....506 Spruce, OALR
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WILLIAMS, M. M. .....Medical Arts Bldg., Op.

SHENANDOAH

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TURTLE CREEK

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UPPER DARBY

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WARREN

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WASHINGTON

McCULLOUGH, CLARENCE JOSEPH....Washington Trust Bldg., ALR
McKENNAN, JAMES W. .....514 Washington Trust Bldg., Op.
McMURRAY, J. B. .....Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C. .....Commercial Bldg., OALR

WILKES-BARRE

BUCKMAN, E. U. .....70 S. Franklin St., OALR
BUCKMAN, LEWIS T. .....83 S. Franklin St., ALR
CARR, GEORGE W. .....54 S. Franklin St., OALR
CONNOLE, JOSEPH V. .....Miners Nat'l Bank Bldg., Op.
KISTLER, O. F. .....43 N. Franklin St., Op.AR
McLAUGHLIN, P. A. .....71 S. Washington St., OALR
MARSDEN, W. C. .....99 N. Franklin St., ALR
SCHAPPERT, N. LOUIS .....59 S. Washington St., OALR

YORK

FACKLER, CHAS. L. .....11 E. Market St., ALR
KLINEDINST, JOHN FRED .....220 S. George St., Op.A

PUERTO RICO

SAN JUAN

FONT, JUAN H. .....Cruz St. No. 13, ALR

RHODE ISLAND

NEWPORT

ALLEN, CARY D. .....U. S. Naval Hospital, ALR
JACOBY, DOUGLAS P. A. .....106 Touro St., Op.AL

PROVIDENCE

ADAMS, FRANK M. .....122 Waterman St., ALR
BOLOTOW, NATHAN A. .....126 Waterman St., ALR
DIMMITT, FRANK W. .....195 Thayer St., OALR
HARVEY, N. DARRELL .....112 Waterman St., OALR
LEECH, J. W. .....167 Angell St., OALR
MESSINGER, H. C. .....210 Angell St., Op.
MUNCY, WM. M. .....162 Angell St., OALR
PORTER, L. B. .....199 Thayer St., OALR
SHARP, BENJ. S. .....339 Thayer St., OALR
TINGLEY, LOUISA PAINE .....171 Westminster St., Op.
WINKLER, HERMAN A. .....224 Thayer St., ALR

SOUTH CAROLINA

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TOWNSEND, JOHN FERRARS .....85 Wentworth St., OALR

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BRISTOW, WALTER J. .....1517 Marion St., ALR

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JERVEY, JAMES W. .....101 Church St., OALR

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SOUTH DAKOTA

HURON

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RAPID CITY

WALSH, JOSEPH MARK .....OALR

SIOUX FALLS

GREGG, JOHN B. .....612 Security Nat'l Bank Bldg., OALR
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JOHNSON, A. EINAR .....Kemp and Broadway, OALR

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HOHF, JULIUS A. .....Clinic Bldg., OALR

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HOGSHEAD, J. M. .....602 Georgia Ave., OALR
LAWWILL, STEWART .....Medical Arts Bldg., OALR
STEELE, WILLARD .....721 Broad St., OALR

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FERGUSON, E. R. .....404 Inman St., ALR

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KNOXVILLE

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LE TELLIER, FORREST S. .....Medical Bldg., Op .
PATTERSON, R. W. .....Medical Arts Bldg., ALR
POTTER, WILLIAM W. P. .....Medical Bldg., OALR
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